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Air Quality Criteria for Particulate Matter and Sulfur Oxides. Volume 1

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The document evaluates and assesses scientific information on the health and welfare effects associated with exposure to various concentrations of sulfur oxides and particulate matter in ambient air. The literature through 1980-81 has been reviewed thoroughly for information relevant to air quality criteria, although the document is not intended as a complete and detailed review of all literature pertaining to sulfur oxides and particulate matter. An attempt has been made to identify the major discrepancies in our current knowledge and understanding of the effects of these pollutants.

Although this document is principally concerned with the health and welfare effects of sulfur oxides and particulate matter, other scientific data are presented and evaluated in order to provide a better understanding of these pollutants in the environment To this end, the document includes chapters that discuss the chemistry and physics of the pollutants; analytical techniques; sources; and types of emissions; environmental concentrations and exposure levels; atmospheric chemistry and dispersion modeling; acidic deposition; effects on vegetation; effects on visibility, climate, and materials; and respiratory, physiological, toxicological, clinical, and epidemiological aspects of human exposure.

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Research Triangle Park, NC 27711

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PREFACE

This document is Volume I of a three-volume revision of Air Quality Criteria for Particulate Matter and Air Quality Criteria for Sulfur Oxides first published in 1969 and 1970, respectively. By law, air quality criteria documents are the basis of the National Ambient Air Quality Standards (NAAQS). The Air Quality Criteria document of which this volume is a part has been prepared in response to specific requirements of Section 108 of the Clean Air Act, as amended in 1977. The Clean Air Act requires that the Administrator of the Environmental Protection Agency periodically review, and as appropriate, update and reissue criteria for NAAQS.

As the legally prescribed basis for deciding on national air quality standards, this document, Air Quality Criteria for Particulate Matter and Sulfur Oxides, delineates health and welfare effects associated with exposure to particulate matter and sulfur oxides and concentrations of those pollutants which cause such effects. The major health and welfare effects of particulate matter and sulfur oxides are discussed in Chapters 8 through 14 in Volume III of the document. To assist the reader in putting the effects into perspective with the real-world environment, Chapters 2 through 7 in Volume II of the document have been prepared and discuss: physical and chemical properties of particulate matter and sulfur oxides; air monitoring and analytical measurement methods; sources and emissions; transport, transformation and fate; and observed ambient concentrations of the pollutants. Also, Chapter 7 in Volume II introduces the reader to the contemporary problem of acidic deposition and potential contributions of sulfur oxides to acidic deposition processes and effects.

This volume, Volume I, introduces the criteria document, explains the rationale behind combining the criteria for particulate matter and sulfur oxides, and briefly summarizes the content of the entire air quality document. However, for a fuller understanding of the health and welfare effects of particulate matter and sulfur oxides, the materials in Volumes II and III of this document should be consulted.

The Agency is pleased to acknowledge the efforts of all persons and groups who have contributed to the preparation of this document. In the last analysis, however, the Environmental Protection Agency accepts full responsibility for its content.

ABSTRACT

The document evaluates and assesses scientific information on the health and welfare effects associated with exposure to various concentrations of sulfur oxides and particulate matter in ambient air. Although the literature through 1980-81 has been reviewed thoroughly for information relevant to air quality criteria, the document is not intended as a complete and detailed review of all literature pertaining to sulfur oxides and particulate matter. An attempt has been made to identify the major discrepancies in our current knowledge and understanding of the effects of these pollutants.

Although this document is principally concerned with the health and welfare effects of sulfur oxides and particulate matter, other scientific data are presented and evaluated in order to provide a better understanding of these pollutants in the environment. To this end, the document includes chapters that discuss the chemistry and physics of the pollutants; analytical techniques; sources; and types of emissions; environmental concentrations and exposure levels; atmospheric chemistry and dispersion modeling; acidic deposition; effects on vegetation; effects on visibility, climate, and materials; and the respiratory, physiological, toxicological, clinical, and epidemiological aspects of human exposure.

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ABBREVIATIONS AND SYMBOLS

Å	Ångström
$A\text{-}aD_{O2}$	Alveolar-arterial difference in partial pressure of oxygen
ACGIH	American Conference of Governmental Industrial Hygienists
ACHEX	Aerosol Characterization Experiment
ACM	Atmospheric Corrosion Monitor
AEC	Atomic Energy Commission
AISI	American Iron and Steel Institute
Al	Aluminum
AM	Alveolar macrophage
AQCD	Air Quality Criteria Document
AQCR	Air Quality Control Region
AQSM	Air Quality Simulation Model
ASTM	American Society for Testing and Materials
ATP	Adenosine triphosphate
BaP	Benzo[a]pyrene
BMRC	British Medical Research Council
BPM	Breaths per minute
BS	British Smokeshade
C	Carbon
Ca^{2+}	Calcium ion
CA	Clean air
CAA	Clean Air Act
CBM	Chlorobenzilidene malonitrile
CAMP	Continuous Air Monitoring Program

CAPITA	Center for Air Pollution Impact and Trend Analysis
CC/TLC%	Closing volume
CH_3O_2	Methyl peroxy radical
CH_3SH	Methyl mercaptan
CHAMP	Community Health Air Monitoring Program
CHESS	Community Health Environmental Surveillance Program
Cl	Chloride ion
CMD	Count mean diameter
CNC	Condensation nuclei counter
CoH	Coefficient of haze
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
COS	Carbonyl sulfide
CS_2	Carbon disulfide
CV	Coefficient of variation; standard deviation divided by the mean
cv	Cultivar
D_{ae}	Aerodynamic equivalent diameter
D_{ar}	Aerodynamic resistance diameter
D_p	Project area diameter
ΔN_2	Delta nitrogen
DMDS	Dimethyl disulfide
DMS	Dimethyl sulfide
D.S.I.R.	Department of Scientific and Industrial Research
EAA	Electrical aerosol analyzer
EAC	Effective area coverage

EDTA	Ethylenediaminetetraacetic acid
EPA	Environmental Protection Agency
EPRI	Electric Power Research Institute
ESCA	Electron spectroscopy for chemical analysis
ET	Extrathoracic
Fe	Iron
Fe_3O_4	Magnetite
FeOOH	Ferric oxyhydroxide
$FeSO_4$	Ferrous sulfate
FEF	Forced expiratory flow
$FEV_{1.0}$	Forced expiratory volume in 1 second
FGD	Flue gas desulfurization
FMC	Fine mass concentration
FPD	Flame-photometric detector
FRC	Functional residual capacity
FVC	Forced expiratory volume/forced vital capacity
GC	Gas chromatography
GRALE	Gamma-ray analysis of light elements
H^+	Hydronium ion; hydrogen ion
Hb	Hemoglobin
H_2CO_3	Carbonic acid
H_2O_2	Hydrogen peroxide
H_2SO_3	Sulfurous acid
H_2SO_4	Sulfuric acid
HCO_3^-	Bicarbonate ion
hi-vol	High-volume

HNO_3	Nitric acid
HO	Hydroxyl radical
HO_2	Peroxy radical
HPLC	High-pressure liquid chromatography
HSO_3^-	Bisulfite ion
ICRP	International Commission on Radiological Protection, Task Group on Lung Dynamics
IFR	Instrument Flight Rules
IP	Inhalable particle
IR	Investigative report
ISP	Interstate Surveillance Program
Kg	Kilogram
K^+	Potassium ion
KPH	Potassium acid phthalate
LC	Lethal concentration
LDH	Lactate dehydrogenase
LEB	Light extinction budget
M	Meter
MAP3S	Multi-state Atmospheric Power Production Pollution Study
MEF	Maximum expiratory flow (pulmonary measurement)
$MEF_{50}VC$	Maximum expiratory flow measured when half the vital capacity has been expelled
MEFR	Maximum expiratory flowrate
mg	Milligram
μg	Microgram
mg/m^3	Milligrams per cubic meter
$\mu g/m^3$	Micrograms per cubic meter

mm	Millimeter
μm	Micrometer
MM	Million metric
MMAD	Mass median aerodynamic diameter
$MMAD_{ar}$	Mass median aerodynamic resistance diameter
MMD	Mass median diameter
MMFR	Maximal mid-expiratory flowrate. Also known as $FEF_{25-75\%}$
MRI	Midwest Research Institute
MTB	Methylthymol blue
MVD	Mean volume diameter
Mg^{2+}	Magnesium ion
$MnCl_2$	Manganese chloride
NAAQS	National Ambient Air Quality Standard
Na^+	Sodium ion
NaCl	Sodium chloride
NAD/NADP	Pyrimidine nucleotides
NADB	National Aerometric Data Bank
NAMS	National Air Monitoring Station
NAPCA	National Air Pollution Control Association
NASN	National Air Surveillance Network
NBS	National Bureau of Standards
NEDS	National Emissions Data System
NH_4^+	Ammonium ion
NH_3	Ammonia
NH_4NO_3	Ammonium nitrate
$(NH_4)_2SO_4$	Ammonium sulfate

NH_4HSO_4	Ammonium bisulfate
NIOSH	National Institute for Occupational Safety and Health
NO_2	Nitrogen dioxide
NO_3^-	Nitrate ion
NPK	Nitrogen, phosphorus, potassium
NRC/NAS	National Research Council/National Academy of Sciences
O_3	Ozone
OBAQI	Observer-based air quality index
ODS	Octadecylsilyl
OECD	Organization for Economic Cooperation and Development
OH	Hydroxyl radical
P	Pulmonary
$PaCO_2$	Partial pressure of carbon dioxide in the arterial blood
PaO_2	Partial pressure of oxygen in the arterial blood
PAH	Polycyclic aromatic hydrocarbons
PbO_2	Lead dioxide
PEFR	Peak expiratory flowrate
PFT	Pulmonary function test
pHa	Arterial pH
PM	Particulate matter
PMT	Photomultiplier tube
POM	Polycyclic organic matter
ppb	Parts per billion
ppm	Parts per million
PVC	Polyvinyl chloride
R_{aw}	Airway resistance (pulmonary measurement)

R_t	Total respiratory flow resistance (pulmonary measurement)
RAPS	Regional Air Pollution Study
RH	Relative humidity
RHC	Reactive hydrocarbons
Rl	Pulmonary flow resistance
RMS	Root mean square
RSD	Recommended site distances
$RSSO_3^-$	Plasma S-sulfonate
RUDS	Reflection unit dirt shade
RV	Residual volume (pulmonary measurement)
S	Sulfur
SAROAD	Storage and Retrieval of Aerometric Data
SEM	Scanning electron microscopy
SES	Socioeconomic status
SG_{aw}	Specific airway conductance
SiO_2	Silicon dioxide
SLAMS	State and Local Air Monitoring Stations
SMSA	Standard metropolitan statistical area
SO_2	Sulfur dioxide
SO_3	Sulfur trioxide ion
SO_3^{2-}	Sulfite ion
SO_4^{2-}	Sulfate ion
SO_x	Sulfur oxides
SPM	Suspended particulate matter
SR_{aw}	Specific airway resistance
SRM	Standard reference material
SSI	Size selective inlet

SURE	Sulfate Regional Experiment
TB	Tracheobronchial
$TB_{\frac{1}{2}}$	Bronchial mucociliary clearance half-time
TCM	Tetrachloromercurate
TGV	Thoracic gas volume
TiO_2	Titanium dioxide
TLC	Total lung capacity
TLV	Threshold limit value
TMR	Total mortality rates
TMTR	Tracheal mucus transport rate
TSP	Total suspended particulate matter
"t" test	Student's statistical test
TV	Tidal volume (pulmonary measurement)
UNAMAP	User's Network for Applied Modeling of Air Pollution
u.v.	Ultraviolet
$\overset{\circ}{V}$	Flowrate during forced expiration (pulmonary measurement)
V_{max} 50% and 75%, etc.	Maximum flowrate calculated at 50 and 75% of expired vital capacity from a partial flow volume curve begun from approximately 60% of inspired vital capacity
VC	Vital capacity (pulmonary measurement)
Ve or V_e	Minute ventilation or minute volume (pulmonary measurement)
VFR	Visual Flight Rules
VISTTA	Visibility Impairment Due to Sulfur Transport and Transformation in the Atmosphere
WHO	World Health Organization
WMO	World Meteorological Organization

WTA	Willingness to accept compensation
WTP	Willingness to pay
ZAPS	Zonal air pollution system

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Chapter 14. Epidemiology Studies on the Effects of Sulfur Oxides and Particulate Matter on Human Health

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1. EXECUTIVE SUMMARY

1.1 INTRODUCTION

1.1.1 Legal Requirements

The purpose of this document is to present air quality criteria for particulate matter and sulfur oxides in accordance with Section 108(a)(2) of the Clean Air Act, 42 U.S.C. §7408(a)(2), which specifies that:

> "Air quality criteria for an air pollutant shall accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the ambient air, in varying quantities. The criteria for an air pollutant, to the extent practicable, shall include information on--
>
> (A) those variable factors (including atmospheric conditions) which of themselves or in combination with other factors may alter the effects on public health or welfare of such air pollutant;
>
> (B) the types of air pollutants which, when present in the atmosphere, may interact with such pollutant to produce an adverse effect on public health or welfare."

National ambient air quality standards are based on such criteria [Clean Air Act Section 109(b), 42 U.S.C. §7409(b)]. Both the criteria and standards are to be reviewed and, as appropriate, revised at five-year intervals beginning not later than December 31, 1980 [Section 109 (d)(1), 42 U.S.C. §7409(d)(1)].

This document constitutes a revision of separate criteria documents previously issued for particulate matter and sulfur oxides (National Air Pollution Control Administration, 1969 and 1970, respectively). A combined document has been prepared for various reasons: (1) Significant amounts of gaseous sulfur dioxide are transformed into particulate sulfate by chemical processes in the atmosphere; (2) It is difficult to separate the relative contributions of sulfur oxides and particulate matter to the mortality and morbidity effects observed in epidemiological studies; (3) Combining the criteria review of the two pollutants, as was done by the World Health Organization, was recommended by the U.S. Environmental Protection Agency's advisory committee on matters related to air quality criteria documents, the Clean Air Scientific Advisory Committee of EPA's Science Advisory Board.

This document describes what is known or anticipated with regard to both the health and welfare effects of particulate matter (PM) and sulfur oxides (SO_x). For purposes of this document, PM is considered to consist of any airborne solid particles and low vapor pressure liquid droplets with an effective diameter smaller than a few hundred micrometers. Important classes of particle sizes within this broad range are identified in subsequent sections of this summary (see Section 1.2, for example). Of the sulfur oxides, only sulfur dioxide (SO_2) occurs at significant concentrations in the atmosphere and is discussed here. Other related sulfur compounds, notably sulfates and sulfuric acid, are covered in the discussion of PM.

With regard to health effects, the document is intended to evaluate the nature and significance of all identifiable effects of PM and SO_x. Under Section 109(b) of the Clean Air Act, the Administrator of EPA is to consider such information in this document in judging which effects are to be considered adverse and to set national primary ambient air quality standards which, based on the criteria and allowing an adequate margin of safety, are requisite to protect the public health. This requires careful assessment of the relationship between levels of exposure to PM and SO_x, via all routes and averaged over appropriate time periods, and biological responses to those exposures. Temporal and spatial distributions of PM and SO_x are considered, as well as such complicating factors as breathing patterns, individual activity levels, special populations of sensitive persons, interactions with other pollutants, and the complex and diverse chemical composition of PM.

The welfare effects to be identified in the criteria document include effects on vegetation, crops, soils, water, animals, manmade materials, weather, visibility, and climate, as well as damage to and deterioration of property, hazards to transportation, and effects on economic values, personal comfort, and well-being [Clean Air Act Section 302(h), 42 U.S.C. §7602(h)]. Under Section 109(b) of the Clean Air Act, the Administrator must consider such information in this document to set national secondary ambient air quality standards that are based on the criteria and are requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such pollutants.

1.1.2 Organization of the Document

This document is being issued in three volumes. The first volume (Volume I) includes Chapter 1 of the document, which contains the general introduction and the executive summary and conclusions for the entire document*; an addendum to the document (discussing certain newly available information on health effects of SO_2) is also included in Volume I, following Chapter 1. Volume II contains Chapters 2 through 7 of the document. Chapters 2 through 5 provide background information on: physical and chemical properties of PM and SO_x; methods for the collection and measurement of such air pollutants; their sources and emissions; and their ambient air concentrations, along with factors affecting exposure of the general population to these pollutants. Chapter 6 evaluates information on atmospheric transport, transformation, and fate of PM and SO_x, followed by an overview discussion in Chapter 7 of potential involvement of PM and SO_x in acidic deposition processes and effects. Volume III contains Chapters 8 through 14 of the document. Chapter 8 evaluates PM and SO_x effects on vegetation, whereas Chapters 9 and 10, respectively, describe effects on visibility and damage to materials attributable to either PM or SO_x. Chapters 11 through 14 evaluate information concerning the health effects of PM and SO_x. More specifically, Chapter 11 discusses respiratory tract deposition of SO_2, sulfur-related particulate matter (especially sulfates), and other types of PM, as well as factors affecting their deposition and biological fate. Chapters 12 and 13 discuss information derived respectively from experimental toxicological studies of animals and from controlled human clinical studies. Chapter 14 discusses epidemiological studies.

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*Note that the second digits of the numerical headings throughout this chapter (Chapter 1) correspond to respective later chapters in the document (e.g., Section 1.2 refers to Chapter 2, Section 1.3 to Chapter 3, etc.).

The extensive literature on PM and SO_x is critically reviewed and evaluated in this document with emphasis on valid studies relevant to the assessment of human health and welfare effects. Air quality information and measurement techniques are discussed in early chapters of the document only to the extent that such information pertains to and helps elucidate the health and welfare effects of PM and SO_x discussed in later chapters. As indicated by the discussion of air quality information, airborne particles of a wide variety of sizes, shapes, and chemical composition are found in the ambient air of the United States in quantities and combinations that vary with time and geographic location. Analysis of the effects of airborne particles is further complicated by complex transformations of various particulate species or their precursor substances during atmospheric transport from sources of emissions that may be hundreds or thousands of kilometers away from humans, other organisms, or materials ultimately exposed to the pollutants. Sulfur dioxide, capable of causing notable health and welfare effects as a gaseous air pollutant, is also the main precursor emitted from manmade sources contributing to the secondary formation of sulfuric acid and sulfate salts. The latter products are in turn major constituents of the PM present as urban aerosols to which large segments of the U.S. population are exposed. Sulfur dioxide and sulfur-related PM species and their associated health and welfare effects are accordingly discussed in considerable detail in the present document. Other individual particulate species of concern, however, are not discussed in as much detail here. Instead, the reader is referred to other EPA air quality criteria or health assessment documents where the effects of such substances are thoroughly reviewed, e.g., _Air Quality Criteria for Lead_ (U. S. Environmental Protection Agency, 1977) and _Air Quality Criteria for Oxides of Nitrogen_ (U. S. Environmental Protection Agency, 1982).

In evaluating available information on the health effects of PM and SO_x in humans, the main focus is on the inhalation of these substances as the most direct and important route of exposure, although it is recognized that some species of PM may cause biological effects via other routes of exposure, such as ingestion or contact with skin. Important issues considered in the document include: (1) patterns of inhalation, deposition, and biological fate of SO_2, sulfur-related PM, and other particulate substances, as a function of their physical and chemical properties; (2) mechanisms of action by which such substances may exert biological effects of potential concern; (3) qualitative characterization of such biological effects; (4) quantitative characterization of dose-response or exposure-effect relationships; and (5) identification of populations at special risk from the effects of PM and SO_x.

In the evaluation of welfare effects of PM and SO_x, consideration is accorded to the direct, acute effects of such substances on visibility, manmade materials, and plant and animal species. Also assessed are the more indirect, long-term effects that might be reasonably anticipated to occur as a consequence of repeated or continuous chronic exposures to low levels of such pollutants. The interactions of PM and SO_x with other factors, such as meteorological variables, and the subsequent deposition of PM and SO_x on and movement through aquatic and terrestrial ecosystems are also addressed.

1.2 PHYSICAL AND CHEMICAL PROPERTIES OF SULFUR OXIDES AND PARTICULATE MATTER

Of the four known gas-phase sulfur oxides (sulfur monoxide, sulfur dioxide, sulfur trioxide, and disulfur monoxide), only sulfur dioxide occurs at significant concentrations in the atmosphere. A colorless gas with pungent odor, SO_2 is emitted from combustion of sulfur-containing fossil fuels, such as coal and oil, as well as from many other sources.

Sulfur dioxide is removed from the atmosphere by gaseous, aqueous, and surface oxidation to form acidic sulfates. Also important are physical removal pathways for SO_2, such as dissolution in raindrops and dry deposition on the earth's surface. Gas-phase oxidation of SO_2 by the hydroxyl (OH) radical is well understood; not so well understood, however, is the oxidation of SO_2 by hydroperoxyl (HO_2) and methylperoxyl (CH_3O_2) radicals. The ready solubility of SO_2 in water is due principally to the formation of bisulfite (HSO_3^-) and sulfite (SO_3^{2-}) ions, which in turn are easily oxidized to form acidic sulfates by reacting with catalytic metal ions and dissolved oxidants. The aqueous-phase oxidation reactions are very complex, and the metal ion catalytic reactions are poorly understood. On the other hand, the oxidation of HSO_3^- and SO_3^{2-} by dissolved hydrogen peroxide (H_2O_2) and ozone (O_3) is well understood; for most situations in the lower troposphere it appears that oxidation by dissolved H_2O_2 is more important than oxidation by dissolved O_3. Sulfur dioxide reacts on the surface of a variety of airborne solid particles, such as ferric oxide, lead dioxide, aluminum oxide, salt, and charcoal. At this time, however, adequate information on the relative importance of the various pathways and on all of the significant reaction rates for SO_2 oxidation in the lower troposphere is not available.

Airborne particles exist in diverse sizes and compositions that can vary widely under the changing influences of source contributions and meteorological conditions. In broad terms, however, airborne particle mass tends to cluster in two principal size groups: coarse particles, generally larger than 2 to 3 micrometers (μm) in diameter; and fine particles, generally smaller than 2 to 3 μm in diameter. The dividing line between the coarse and the fine sizes is frequently given as 2.5 μm, but the dividing line according to chemical composition is neither sharp nor fixed; it can depend on the contributing sources, on meteorology, and on the age of the aerosol. The curves in Figure 1-1 represent the influence of these parameters.

Fine particle volume (or mass) distributions may exhibit two modes. Those particles in the nuclei mode (which includes particles from 0.005 to 0.05 μm in diameter) form near sources by condensation of vapors produced by high temperature processes such as fossil-fuel combustion. Particles in the accumulation mode (which includes particles from 0.05 to about 2 μm in diameter) form principally by coagulation or growth through vapor condensation of the short-lived particles in the nuclei mode. Typically, 80 percent or more of the atmospheric

Figure 1-1. Idealized size distributions for atmospheric particles under various conditions.

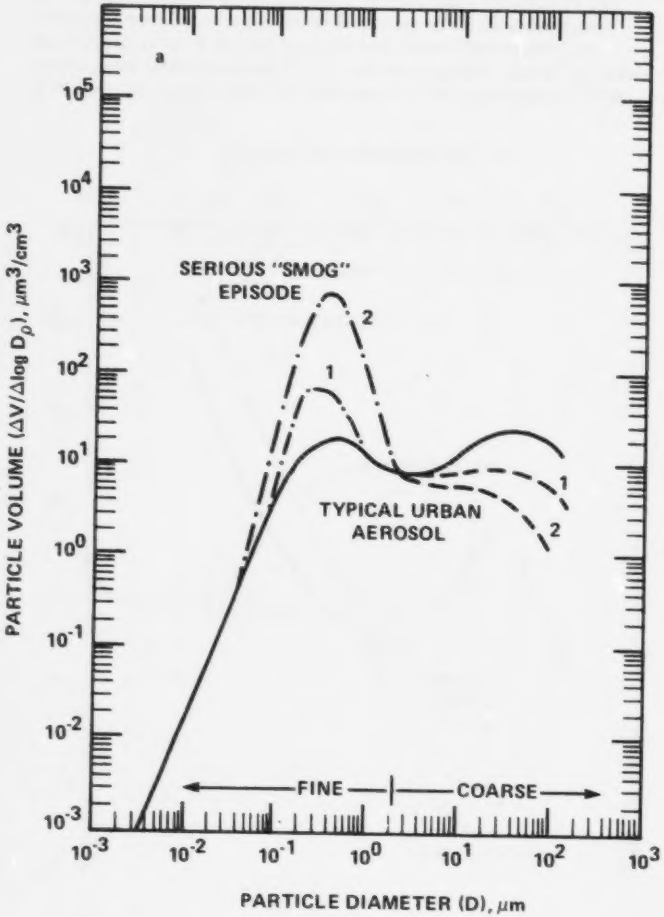

Figure 1-1a. Idealized size distribution for particles found in typical urban aerosols (mainly from anthropogenic sources) under varying weather conditions. Note bimodal distribution under usual conditions and shift in distribution (increasing fine-mode particles, decreasing coarse-mode particles) under stagnation (1) and serious "smog" conditions (2), respectively.

Source: Adapted from Slinn (1976).

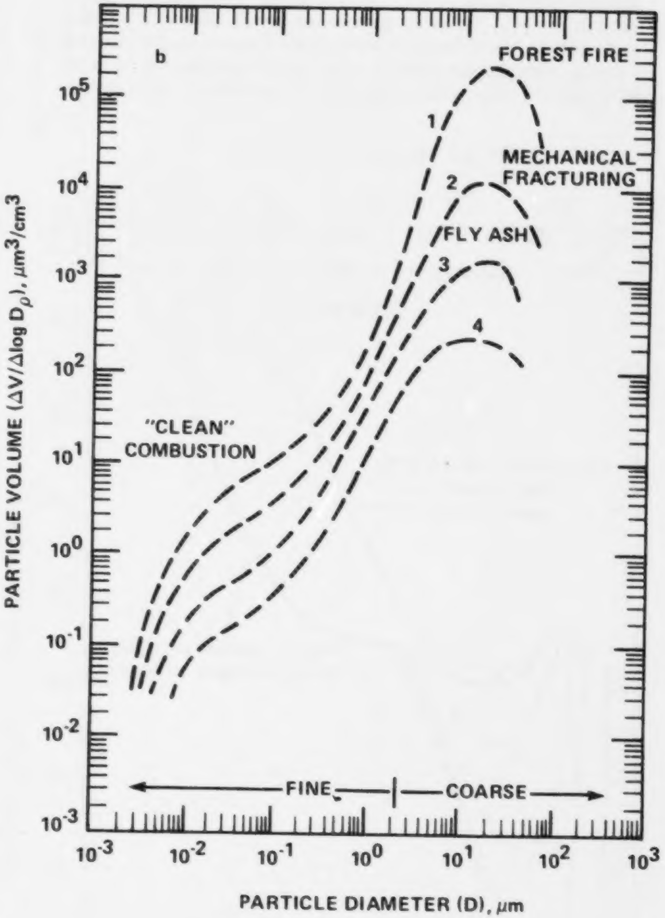

Figure 1-1b. Idealized size distribution for atmospheric particles from anthropogenic sources, showing fine particle contributions from "clean" high-temperature combustion and coarse particle contributions from "dirty" fly ash sources, forest fires, and crushing and grinding operations. Note change in distribution near sources (1) and at increasing distances (2,3,4) from sources.

Source: Adapted from Slinn (1976).

Figure 1-1. (Continued)

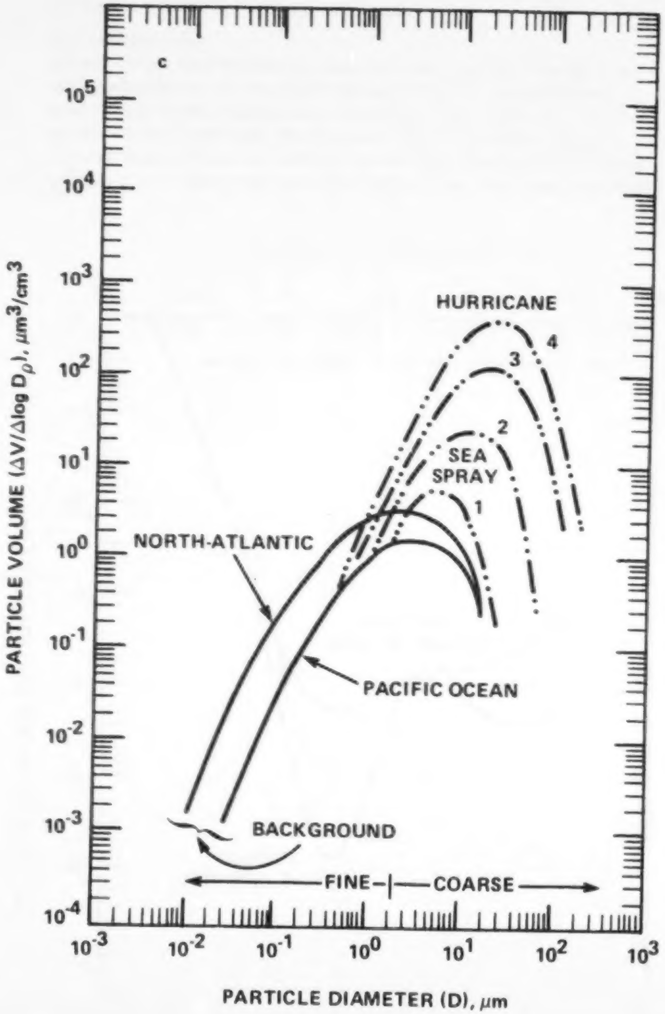

Figure 1-1c. Idealized size distribution for atmospheric particles from natural sources in a marine setting. Note, in comparison to typical background levels over open ocean, increasing levels of coarse-mode particles ranging from those found in sea spray (1,2) to the extreme cases of storms (3) and hurricanes (4).

Source: Adapted from Slinn (1976).

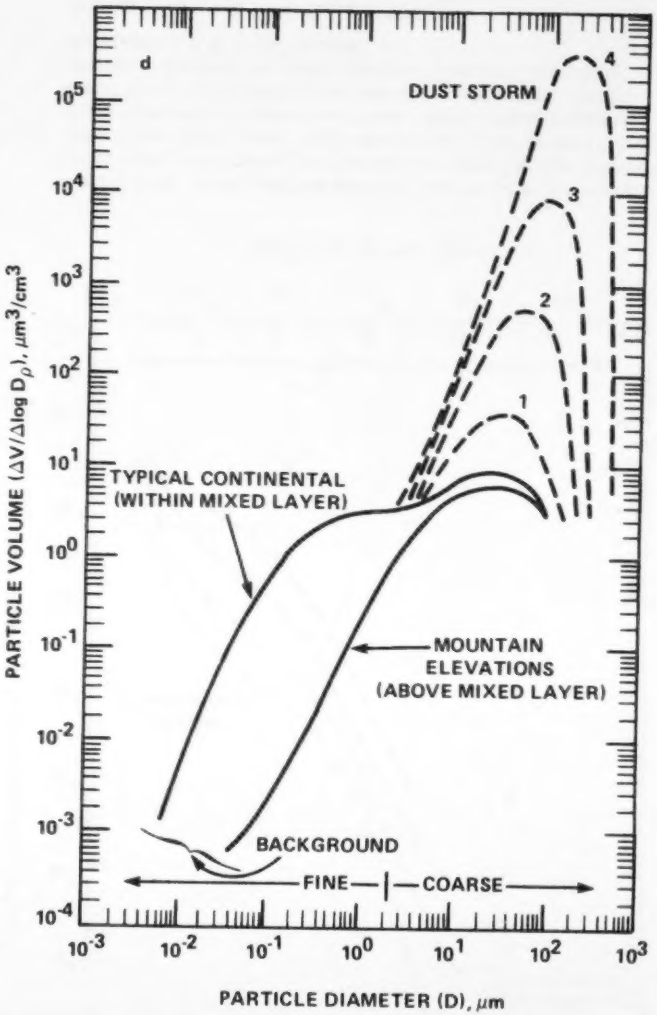

Figure 1-1d. Idealized size distribution for atmospheric particles from natural sources in a continental setting. Note, in comparison to usual background profiles over typical continental and high-elevation mountain areas, increasing contributions of coarse-mode particles from wind-blown dusts (1,2,3), ranging to the extreme case of a dust storm (4).

Source: Adapted from Slinn (1976).

sulfate mass occurs in the accumulation mode. Accumulation-mode particles normally do not grow into the coarse mode. Coarse particles include re-entrained surface dust, salt spray, and particles formed by mechanical processes such as grinding.

Primary particles are directly discharged from manmade or natural sources. Secondary particles form by chemical and physical reactions in the atmosphere, and most of the reactants involved are emitted to the air as gaseous pollutants.

In the atmosphere, particle growth and chemical transformation occur through gas-particle and particle-particle interactions. Gas-particle interactions include condensation of low vapor pressure molecules, such as sulfuric acid (H_2SO_4) and organic compounds; such condensation occurs principally on fine particles. The only particle-particle interaction important in atmospheric processes is coagulation among fine particles.

As shown in Figure 1-2, major components of fine atmospheric particles include sulfates, carbonaceous material, ammonium, lead, and nitrate. Coarse particles consist mainly of oxides of silicon, aluminum, calcium, and iron, as well as calcium carbonate, sea salt, and material such as tire particles and vegetation-related particles (e.g., pollen, spores). Note that the distributions of fine and coarse particles overlap and that some chemical species found predominantly in one mode may also be found in the other mode.

The carbonaceous component of fine particles contains both elemental carbon (graphite and soot) and nonvolatile organic carbon (hydrocarbons emitted in combustion exhaust and secondary organics formed by photochemistry). In many urban and nonurban areas, these species may be the most abundant fine particles after sulfates. Secondary organic particles form by oxidation of primary organics by a cycle that also involves ozone and nitrogen oxides. Atmospheric reactions of nitrogen oxides yield nitric acid vapor (HNO_3) that may accumulate as nitrate particles in the fine and coarse modes. Details of the chemical pathways for forming nitrate particles and secondary organics are not well established, and the validity of historical nitrate data is questionable.

Most atmospheric sulfates and nitrates are water-soluble and have a tendency to absorb moisture. Hygroscopic growth of sulfate-containing particles has a profound effect on their size, reactivity, and other physical properties which in turn influence their biological and physical effects.

1.3 TECHNIQUES FOR COLLECTION AND ANALYSIS OF PARTICULATE MATTER AND SULFUR OXIDES

Various instruments are used to measure levels of particulate matter and sulfur oxides. The instruments used in laboratory studies of the effects of PM and SO_x may differ greatly from those used to monitor ambient air levels. Differences in exposure characterization obtained from these various methods may have important implications for the derivation of quantitative dose-response relationships from different types of studies. Ambient air monitoring methods are most important for epidemiological studies on the health effects of PM and SO_x and for assessing compliance with related NAAQS; such monitoring methods are, therefore, considered in detail in Chapter 3.

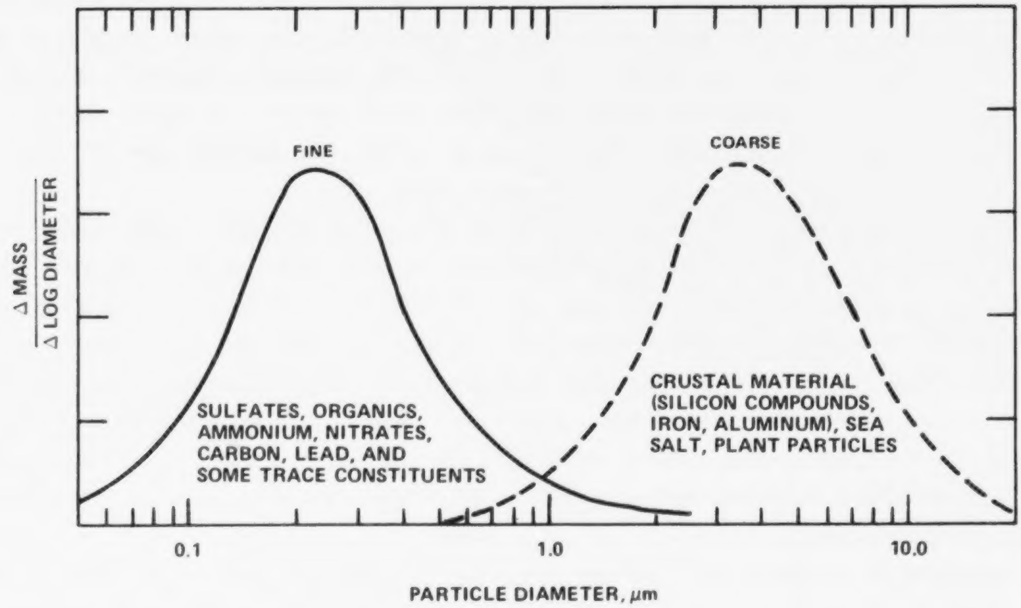

Figure 1-2. Idealized representation of typical fine- and coarse-particle mass and chemical composition distribution in an urban aerosol. Although some overlap exists, note substantial differences in chemical composition of fine versus coarse modes. Chemical species of each mode are listed in approximate order of relative mass contribution. Note that the ordinate is linear and not logarithmic.

1.3.1 Summary of Sulfur Dioxide Measurement Techniques

Methods for the measurement of SO_2 can be classified as: (1) manual methods, which involve collection of the sample over a specified time period and subsequent analysis by a variety of analytical techniques, or (2) automated methods, in which sample collection and analysis are performed continuously and automatically.

In the commonly used manual methods, the techniques for the analysis of the collected sample are based on colorimetric, titrimetric, turbidimetric, gravimetric, x-ray fluorescent, chemiluminescent, and ion exchange chromatographic measurement principles.

The most widely used manual method for the determination of atmospheric SO_2 is the pararosaniline method developed by West and Gaeke. An improved version of this colorimetric method, adopted as the EPA reference method in 1971, is capable of measuring ambient SO_2 concentrations as low as 25 $\mu g/m^3$ (0.01 ppm) with sampling times ranging from 30 minutes to 24 hours. The method has acceptable specificity for SO_2, if properly implemented to minimize interference by nitrogen dioxide or metal oxides; but samples collected in tetrachloromercurate (II) are subject to a temperature-dependent decay which can result in an underestimation of the ambient SO_2 concentration. Temperature control during sample collection, shipment, and storage effectively minimizes this decay problem. A recent variation of the pararosaniline method uses a buffered formaldehyde solution for sample collection and is reported to be less susceptible to the temperature-dependent decay problem. Some American epidemiological studies employed the West-Gaeke method for measurement of SO_2 concentrations to assess possible health effects of SO_2.

A titrimetric method based on collection of SO_2 in dilute hydrogen peroxide, followed by titration of the resultant H_2SO_4 with standard alkali, is the standard method used extensively in Great Britain. Although simple to perform, the method requires long sampling times (24 hours) and is subject to interference from atmospheric acids and bases. Additional sources of error include evaporation of reagent during sampling, titration errors, and alkaline contamination of glassware. The hydrogen peroxide method was also adopted as a standard method by the Organization for Economic Cooperation and Development (OECD) and was employed to provide aerometric SO_2 estimates reported in many British and European epidemiological studies.

Methods that employ alkali-impregnated filter papers for the collection of SO_2 and subsequent analysis as sulfite or sulfate have also been developed. Most of these methods involve an extraction step prior to analysis, although nondispersive x-ray fluorescence has been used for the direct measurement of SO_2 collected on sodium carbonate-impregnated membrane filters. These methods, however, have not yet found wide-spread use in the United States for routine ambient air monitoring purposes or in generating SO_2 aerometric data used in epidemiological studies of the health effects of SO_2.

Two of the most sensitive methods now available for measurement of SO_2 use principles based on chemiluminescence and ion exchange chromatography. In the chemiluminescence method, SO_2 is absorbed in a tetrachloromercurate solution and subsequently oxidized with potassium permanganate. The oxidation of the absorbed SO_2 is accompanied by a chemiluminescence that is detected by a photomultiplier tube. One method uses ion exchange chromatography to determine ambient levels of SO_2 which have been absorbed into dilute hydrogen peroxide and oxidized to sulfate. Another ion chromatographic approach using a buffered formaldehyde absorbing reagent has also been reported. These methods, however, have not yet been widely employed for routine monitoring or other field uses.

Sulfation methods, based on reaction of airborne sulfur compounds with lead dioxide paste to form lead sulfate, have commonly been used to estimate ambient SO_2 concentrations over extended time periods. However, the accuracy of sulfation methods is subject to many physical and chemical variables and other interferences (such as wind speed, temperature, and humidity). Moreover, the method is not specific for SO_2, since it is affected by other sulfur compounds (such as sulfates) as well. Thus, although sulfation rate (mg SO_3/100 cm^2/day) is commonly converted to a rough estimate of SO_2 concentration (in ppm) by multiplying the sulfation rate by the factor 0.03, this cannot be accepted as an accurate measure of atmospheric SO_2 concentrations. This fact is important in view of the past widespread use of lead dioxide gauges in the United Kingdom as the basis for aerometric SO_2 data reported in some pre-1960s British epidemiological studies. Also, sulfation-rate methods were used in some American epidemiological studies, as noted in Section 1.14.

Automated methods for measurement of ambient levels of sulfur dioxide have gained widespread use in the air-monitoring community. Certain of the earliest continuous SO_2 analyzers were based on conductivity and coulometry. These first generation analyzers were subject to interference by a wide variety of substances present in typical ambient atmospheres. However, more recent commercially available analyzers using these measurement principles exhibit improved specificity for SO_2 through the incorporation of sophisticated chemical and physical scrubbers. Early continuous colorimetric analyzers using West-Gaeke type reagents and having good sensitivity and acceptable specificity for SO_2 were fraught with various mechanical problems, required frequent calibration, and thus never gained widespread acceptance.

Continuous sulfur dioxide analyzers using the techniques of flame photometric detection (FPD), fluorescence, and second-derivative spectrometry have been developed over the past 10 years and are commercially available from a number of air monitoring instrumentation companies. Flame photometric detection of ambient SO_2 is based on measurement of the band emission of excited S_2^* molecules formed from sulfur species in a hydrogen-rich flame. The FPD analyzers exhibit high sensitivity and fast response, but must be used with selective scrubbers or coupled with gas chromatographs when high specificity is required.

Fluorescence analyzers are based on detection of the characteristic fluorescence of the SO_2 molecule when it is irradiated by UV light. These analyzers have acceptable sensitivity and response times, are insensitive to sample flowrate, and require no support gases. They are subject to interference by water vapor (due to quenching effects) and certain aromatic hydrocarbons, and therefore must incorporate ways to minimize these species or their effects.

Second-derivative spectrometry is a highly specific technique for measurement of SO_2 in the air, and continuous analyzers based on this principle are commercially available. The analyzers are insensitive to sample flowrate and require no support gases, but relatively high sample flowrates are required to achieve reasonable response times. Excessive electronic noise and inherent lack of precision can be problems with these analyzers.

Continuous analyzers based on many of the above measurement principles (conductivity, coulometry, flame photometry, fluorescence, and second-derivative spectrometry) have been designated by EPA as equivalent methods for the measurement of SO_2 in the atmosphere. Testing of these analyzers by the manufacturers prior to designation has demonstrated adequate performance for use when an EPA reference or equivalent method is desired or required. Testing of these methods by EPA has verified their performance and has also demonstrated excellent comparability among these designated methods under typical monitoring conditions.

1.3.2 Summary of Measurement Techniques for Particulate Matter

Sampling particulate matter suspended in ambient air presents a complex task because of the spectrum of particle sizes and shapes. Separating particles by aerodynamic size provides a simplification by disregarding variations in particle shape and relying on particle settling velocity. Note that the aerodynamic diameter of a particle is not a direct measurement of its size but is the equivalent diameter of a spherical particle of specific gravity which would settle at the same rate as the particle in question. Samplers can be designed to collect particles within sharply defined ranges of aerodynamic diameters or to simulate the deposition pattern of particles in the human respiratory system, which exhibits a more gradual transition from acceptance to exclusion of particles. High-volume (hi-vol) samplers with selective inlets, dichotomous samplers, cascade impactors, and cyclone samplers are the most common devices with specifically designed collection characteristics. Carefully collected size distributions of ambient particle mass have shown that most particle samplers underestimate the concentration of particles in the air because of sensitivity to external factors such as wind speed or because of internal particle losses.

Mass concentrations can be estimated using methods that measure an integral property of particles such as optical reflectance. Empirical relationships between mass concentrations and the integral measurement have been developed and can be used to predict mass concentration. However, without a valid physical model relating to the measurements, plus empirical data to demonstrate the model, these techniques have a limited ability to estimate mass concentrations. These conditions are poorly met in the case of reflectance or transmission tape samplers, fairly well met in the integrating nephelometer, and very well met in the case of beta-ray attenuation analysis.

Sampling accuracy can be estimated through key sampling components, such as flowrate and inlet sampling effectiveness. These component measurements provide a means of intercomparing methods, even though a reference measurement technique is not available. Recent interest in larger particle sampler cutpoints (e.g., 15 μm) have resulted in wind tunnel test procedures that determine sampling effectiveness of particle samplers under controlled conditions. Such measurements have added significantly to the ability to estimate particle sampling accuracy.

The hi-vol sampler collects particles on a glass-fiber filter by drawing air through the filter at a flowrate of approximately 1.5 m^3/min, thus sampling a higher volume of air per unit of time than the above sampling methods for PM. The hi-vol sampler is widely used in the United States to measure what is known as "total suspended particulate matter" (TSP). Recent evaluations show that the hi-vol sampler has cutpoints of ≅25 μm at a wind speed of 24 kph and 45 μm at 2 kph. Although the sampling effectiveness is wind-speed sensitive, wind speed is estimated to produce no more than a 10-percent day-to-day variability for the same ambient concentration for typical conditions. The hi-vol is one of the most reproducible particle samplers currently in use, with a typical coefficient of variation of 3 to 5 percent. A significant problem associated with the glass-fiber filter used on the hi-vol is the formation of artifact mass caused by the presence of acid gases in the air (artifactual formation of sulfates from SO_2 being one example). These artifacts can add 6 to 7 μg/m^3 to a 24-hour sample. The hi-vol sampler has been extensively used in the United States for routine monitoring purposes and has provided estimates of total suspended particulate (TSP) mass used in many American epidemiological studies of the health effects of PM.

The dichotomous sampler was designed to collect the fine and coarse ambient particle fractions, typically providing a separation at 2.5 μm. This sampler uses Teflon® filters to minimize artifact mass formation and is available in versions for manual or automatic field operation. The earlier inlets used with this sampler were very wind-speed dependent, but newer versions are much improved. Because of low sampling flowrate, the dichotomous sampler collects submilligram quantities of particles and requires microbalance analyses, but is capable of reproducibilities of ±10 percent or better. The method, however, has only recently begun to be evaluated for possible routine field use and has not yet been extensively employed for generating size-selective data on PM mass in relation to health effects evaluated in epidemiological studies.

Cyclone samplers with cutpoints in the vicinity of 2 μm have been used for years to separate the fine particle fraction. A version is also available for personal dosimeter sampling. Cyclone samplers can be designed to cover a range of sampling flowrates and are available in a variety of physical sizes. Applications of cyclone samplers are found in 10- and 15-μm cutpoint inlets for the dichotomous sampler. Cyclone sampling systems could be expected to have coefficients of variations similar to that of the dichotomous sampler and have also found only limited use until recently in epidemiological studies of PM health effects.

The Size Selective Inlet (SSI) hi-vol collects samples containing particles less than 15 μm for comparison with TSP. Except for the inlet, this sampler is identical to the TSP hi-vol. It is expected to have the same basic characteristics and is presently being evaluated for possible routine monitoring use in the field.

Cascade impactors have been used extensively to obtain mass distribution by particle size. Because care must be exercised to prevent errors, such as those caused by particle bounce between stages, these samplers are normally not operated as routine monitors. A study by Miller and DeKoning (1974) comparing cascade impactors with hi-vol samplers showed inconsistencies in the mass median diameter and total mass collections of the impactors.

Samplers that derive mass concentrations by analytical techniques other than direct weight have been used extensively. One of the earliest was the British smokeshade (BS) sampler, which measures the reflectance of particles collected on a filter and uses empirical relationships to predict mass concentration. These relationships have been shown by Bailey and Clayton (1980) to be more sensitive to carbon concentrations than mass, and hence are very difficult to interpret as either total or size-selective PM mass present in the atmosphere. More specifically, the BS method and its standard variations typically collect PM with an ≅4.5 μm D_{50} cutpoint under field conditions (McFarland et al., 1982). Thus, regardless of whether larger particles are present in the atmosphere, the BS method collects predominantly small particles. The BS method neither directly measures mass nor determines chemical composition of collected PM. Rather, it measures light absorption of particles as indicated by reflectance from a stain formed by the particles collected on filter paper, which is somewhat inefficient for collecting very fine particles. The reflectance of light from the stain depends both on the density of the stain, or amount of PM collected, and the optical properties of the collected PM. Smoke particles composed of elemental carbon found in incomplete fossil-fuel combustion products typically make the greatest contribution to darkness of the stain, especially in urban areas. Thus, the amount of elemental carbon, but not organic carbon, present in the stain tends to be most highly correlated with BS reflectance readings. Other nonblack, noncarbon particles also have optical properties such that they can affect the reflectance readings, although their contribution to optical absorption is usually negligible.

Since the relative proportions of atmospheric carbon and noncarbon PM can vary greatly from site to site or from one time to another at the same site, the same absolute BS reflectance reading can be associated with markedly different amounts (or mass) of collected particles or, in unusual circumstances, even with markedly different amounts of carbon. Site-specific calibrations of reflectance readings against actual mass measurements obtained by collocated gravimetric monitoring devices are therefore necessary to obtain estimates of atmospheric concentrations of particulate matter based on the BS method. A single calibration curve relating mass or atmospheric concentration (in $\mu g/m^3$) of particulate matter to BS reflectance readings obtained at a given site may serve as a basis for crude estimates of the levels of PM (mainly small particles) at that site over time, so long as the chemical composition and relative proportions of elemental carbon and noncarbon PM do not change substantially. However, the actual mass or smoke concentrations present at a particular site may differ markedly from the values calculated from a given reflectance reading on either of the two most widely used standard curves (the British and OECD standard smoke curves). Thus, great care must be taken in interpreting the meaning of any BS value reported in terms of $\mu g/m^3$, especially as employed in many of the British and European epidemiological studies discussed in Chapter 14.

The AISI light transmittance method is similar in approach to the BS technique and has been employed for routine monitoring in some American cities. The instrument collects particles with a D_{50} cutpoint of ≅5.0 μm aerodynamic diameter and uses an air intake similar to that of the BS method. Particulate matter collects on a filter-paper tape that is periodically advanced to allow accumulation of another stain. Opacity of the stain is determined by transmittance of light through the deposited material and the tape, with results expressed in terms of optical density or coefficient of haze (CoH) units per 1000 linear feet of air sampled (rather than mass units). Readings in CoH units are somewhat more responsive to noncarbon particles than are BS measurements; but, again, the AISI method does not directly measure mass or determine chemical composition of the PM collected. Any attempt to relate CoHs to $\mu g/m^3$ would require site-specific calibration of CoH readings against mass measurements determined by a collocated gravimetric device, but the accuracy of such mass estimates could still be subject to question. This type of calibration, however, has only been attempted for New York City and has only very limited possible applicability for certain New York City aerometric data reported in some epidemiological studies.

Regan et al. (1979) showed that this sampler correlates favorably with gravimetric measurements limited to the smaller particle sizes. Waggoner and Weiss (1980) and Groblicki et al. (1980) also reported good correlation between the integrating nephelometer and gravimetric fine particle mass. The Electrical Aerosol Analyzer (EAA), however, was shown to have difficulties in reliably predicting gravimetric mass measurements (Mulholland et al., 1980). These latter methods, unlike the AISI method, have not been used in gathering PM data used in epidemiological studies; but the nephelometer has yielded information useful in quantifying the effects of fine-mode PM on visibility (see Section 1.9).

Since the hi-vol method collects particles considerably larger than those collected by the BS or AISI methods, intercomparisons or conversions of PM measurements by the BS or AISI methods to equivalent TSP units, or vice versa, are severely limited. For example, as shown by several studies, no consistent relationship exists between BS and TSP measurements taken at various sites or even at the same site during various seasons. One exception appears to be the relationship between BS and TSP observed during severe London air pollution episodes when low wind-speed conditions resulted in settling out of larger coarse-mode particles. Since fine-mode particles consequently predominated, TSP and BS levels (in excess of about 500 $\mu g/m^3$) tended to converge, as would be expected if only fine-mode particles were present.

Optical particle morphology techniques are very useful for identifying the character and sources of collected particles. Bradway et al. (1976), however, noted that these techniques are dependent on the skill of the microscopist and stressed the need for careful quality assurance procedures. In general, such methods have not found wide-spread use beyond highly specialized research applications.

An extensive list of analytical techniques is available to determine chemical properties of particles collected on a suitable substrate. Many of the analytical techniques, such as those for elemental sulfur, have been demonstrated (Camp et al., 1978) to be more precise than the analyses for gravimetric mass concentration. Methods are available to provide reliable analyses for sulfates, nitrates, organic fractions, and elemental composition (e.g., sulfur, lead, silicon). Not all analyses can be performed on all particle samples because of factors such as incompatible substrates and inadequate sample size. Misinterpretation of analytical results can occur when samples have not been appropriately segregated by particle size and when artifact mass is formed on the substrate rather than collected in particulate form. Positive artifacts are particularly likely in sulfate and nitrate determinations, and negative nitrate artifacts also occur.

Sampling technology is available to meet specific requirements such as providing sharp cutpoints, cutpoints that match particle deposition models, separate collection of fine and coarse particles, automated sample collection capability, collection of at least milligram quantities of particles, minimal interaction of the substrate with the collected particles, ability to produce particle size distribution data, low purchase cost, and simple operating procedures. Not all of these sampling requirements may be needed for a measurement study. Currently, there is no single sampler which meets all requirements, but samplers are available to meet most typical requirements if the overall accuracy and reproducibility of the method are consistent with the objectives of a study.

1.4 SOURCES AND EMISSIONS OF PARTICULATE MATTER AND SULFUR OXIDES

Both natural and manmade sources emit particulate matter and sulfur oxides into the atmosphere. Natural particulate emissions include dust, sea spray, volcanic emissions, biogenic emanations (e.g., from plants), and emissions from wildfires. Manmade emissions originate from stationary point sources, fugitive sources (such as roadway and industrial dust), and transportation sources (vehicle exhausts). See Section 1.2 for information regarding physical (e.g., size) and chemical properties of PM emitted from these different sources.

Reliable estimates for natural emissions of PM and SO_x specific to the United States are not available. Proportional interpolations from global estimates indicate that in the United States natural sources may emit 84 million metric tons of particles yearly ; estimates of biogenic sulfur emissions in the United States suggest a total in the range of 0.2 to 0.5 million metric tons annually. Additional contributions from coastal and oceanic sources may also be significant. In contrast, manmade sources are estimated to emit 125 million to 385 million metric tons of PM and 27 million metric tons of SO_x (mostly SO_2) per year in the United States. However, these numbers should be considered no more than rough estimates because of the assumptions and crude approximations inherent in most emissions calculations.

The proximity of emissions to humans often is more important than relative intensity. For example, emissions from combustion of home-heating fuels and transportation sources are minor on a national level. However, because they are emitted in densely populated areas and close to ground level, the possibility of effects on human health and welfare is thereby greatly increased. On the other hand, dust from unpaved roads appears to be significant, but usually occurs in rural areas, and tends to settle out quickly, lessening any possible consequences. Conversely, although some natural source emissions can be fairly intense (volcanic ash or sulfur from marshlands, for example), in general their effects are lessened because they tend to be distributed fairly broadly nationwide. Consequently, simple comparisons of total national tonnages of manmade versus natural emissions will seldom reflect the impact that localized manmade sources can have on an area's air quality. For such reasons, certain manmade sources, particularly stationary point sources, have been given special attention in this document. Historical trends in anthropogenic emissions of PM (excluding fugitive emissions) and SO_x are shown in Table 1-1.

Most manmade sulfur oxide emissions come from stationary point sources, and more than 90 percent of these discharges are in the form of SO_2. The balance consists of sulfates. Most natural sulfur is emitted as reduced sulfur compounds, some portion of which probably become oxidized in the atmosphere to SO_2 and sulfates.

Characteristics of particle emissions vary with the source and a host of other factors. Primary particles from natural sources tend to be coarse. About 50 percent are larger than 10 μm. Particles from nonindustrial fugitive sources, such as unpaved roads and wind-eroded farmland, are significant on a mass basis, constituting an estimated 110 to 370 million metric tons a year. However, only about 20 percent of this particulate matter is less than 1 μm in size. On the other hand, most particles emitted by stationary and transportation sources are less than 2.5 μm in diameter. In addition, the variety of different toxic elements found in fine material from stationary point sources tends to exceed that typically found in emissions from manmade or natural fugitive sources.

Fugitive dust emissions exceed those from stationary point sources in most Air Quality Control Regions having high TSP loadings. However, the impact of this pollution on populated areas may be lessened because: (1) a major portion of these emissions consists of large particles that settle out in a short distance, and (2) most sources, such as unpaved roads, exist in rural areas and their emissions spread over areas with low population densities.

TABLE 1-1. NATIONAL ESTIMATES OF PARTICULATE AND SULFUR OXIDE EMISSIONS

(a). PARTICULATE EMISSIONS[a]
(10^6 metric tons per year)

SOURCE CATEGORY	1940	1950	1960	1970	1975	1978
Stationary fuel combustion	8.7	8.1	6.7	7.2	5.1	3.8
Industrial processes	9.9	12.6	14.1	12.8	7.4	6.2
Solid waste disposal	0.5	0.7	0.9	1.1	0.5	0.5
Transportation	0.5	1.1	0.6	1.1	1.0	1.3
Miscellaneous[b]	5.2	3.7	3.3	1.0	0.6	0.7
TOTAL	24.8	26.2	25.6	23.2	14.6	12.5

(b). SULFUR OXIDE EMISSIONS
(10^6 metric tons per year)

SOURCE CATEGORY	1940	1950	1960	1970	1975	1978
Stationary fuel combustion	15.1	16.6	15.7	22.7	20.9	22.1
Industrial processes	3.4	4.1	4.8	6.2	4.5	4.1
Solid waste disposal	0.0	0.1	0.0	0.1	0.0	0.0
Transportation	0.6	0.8	0.5	0.7	0.8	0.8
Miscellaneous[b]	0.4	0.4	0.4	0.1	0.0	0.0
TOTAL	19.5	22.0	21.4	29.8	26.2	27.0

[a] Table does not include industrial-process fugitive particulate emissions, and non-industrial fugitive emissions from paved and unpaved roads, wind erosion, construction activities, agricultural tilling, and mining activities.

[b] Table includes forest fires, agricultural burning, coal-refuse burning, and structural fires.

Source: U.S. Environmental Protection Agency (1978, 1980)

1.5 CONCENTRATIONS AND EXPOSURE

Sulfur oxide concentrations in the air have been markedly reduced during the past 15 years by restrictions on sulfur content in fuels, control devices on stationary and other major sources, and tall stacks that disperse power plant exhausts. Currently, only 1 percent of the SO_2 monitoring sites show annual levels above 80 $\mu g/m^3$ (0.03 ppm), as compared with 16 percent of the monitoring stations that reported annual means above this level in 1970. Despite this change, some areas still report high short-term SO_2 concentrations (see areas indicated in Figure 1-3). Hourly values of 4000 to 6000 $\mu g/m^3$ (1.5 to 2.3 ppm) are common near large smelters. Maximum hourly values above 1000 $\mu g/m^3$ (0.4 ppm) exist in about 100 locations in the United States. Near isolated point sources, such peaks may be reached very rapidly and be of only short duration (see, for example, Figure 1-4).

Following a downward trend between 1970 and 1974, TSP concentrations have not changed significantly in recent years. Dusty, arid parts of the country still have somewhat elevated TSP values, as do industrialized cities in the East and Far West. Ninetieth percentile values (values exceeded 10 percent of the time) of 24-hr TSP $\geq$85 $\mu g/m^3$ are reported in every part of the United States except Alaska. Annual mean TSP values generally range from 55 $\mu g/m^3$ in New England to 100 $\mu g/m^3$ in the arid Southwest.

As discussed in Section 1.2, particulate matter is generally distributed in fine- and coarse-mode size ranges of differing chemical compositions. A comparison of dichotomous sampler data (fine and coarse) and hi-vol particle measurements (TSP) for selected urban, suburban, and rural sites is shown in Figure 1-5. The figure suggests a seasonal pattern of high summer and low winter concentrations that is most evident for fine particles. Fine particles typically contribute about one-third of TSP mass in urban areas. The sulfate ion usually accounts for about 40 percent of the fine-mode mass; sulfate compounds collectively account for the majority of the fine-mode mass. Large areas of the United States experience 10 $\mu g/m^3$ or greater sulfate levels for one or two periods of a month or more every year. These areas are so large that no background levels of fine particles can now be measured east of the Mississippi River. Southern California experiences high levels of sulfates and nitrates, particularly during photochemical smog incidents. Extremely high levels (>100 $\mu g/m^3$) of organic aerosols also occur in this area, particularly during afternoon periods of intensive ozone formation. These organic aerosols consist largely of dicarboxylic acids and other polyfunctional compounds. Concentrations of toxic organic particulate matter and trace metals are highest in cities. Levels of some fine-particle components have decreased because of control measures, such as reduction of lead in gasoline.

Coarse particles tend to settle close to sources. In most cases, these particles account for two-thirds of the TSP mass. During the summer, in dry regions such as Phoenix, Oklahoma City, El Paso, and Denver, they may contribute even higher proportions. The primary cause of high TSP appears to be local dust; but, in industrialized cities, evidence exists for significant contributions of soot, fly ash, and industrial fugitive emissions.

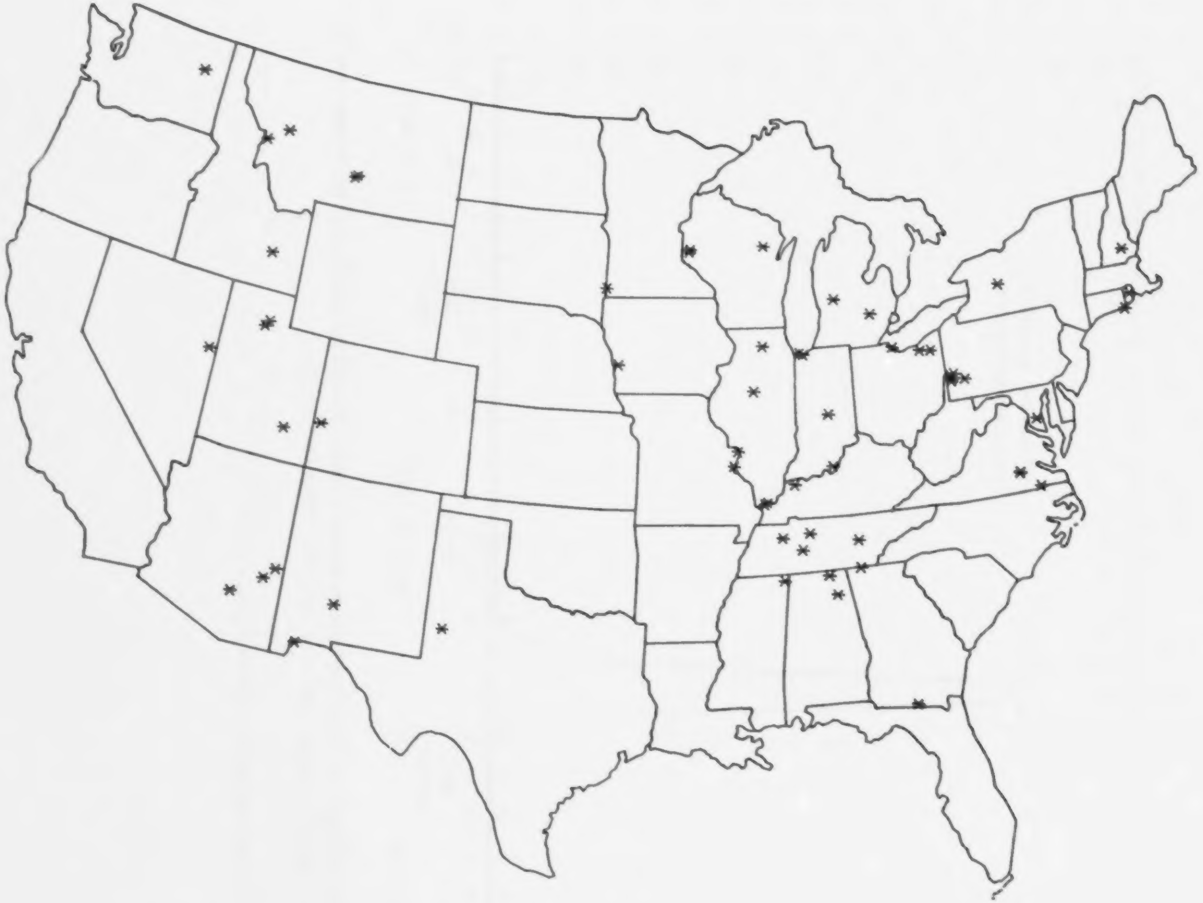

Figure 1-3. Characterization of 1974-76 national SO_2 status is shown by second highest 24-hr. average concentration. Asterisks denote counties for which this level exceeded 365 $\mu g/m^3$. (The current 24-hr. primary standard is 365 $\mu g/m^3$, which is not to be exceeded more than once per year. Alaska and Hawaii reported no such exceedences.)

Source: Monitoring and Reports Branch, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency.

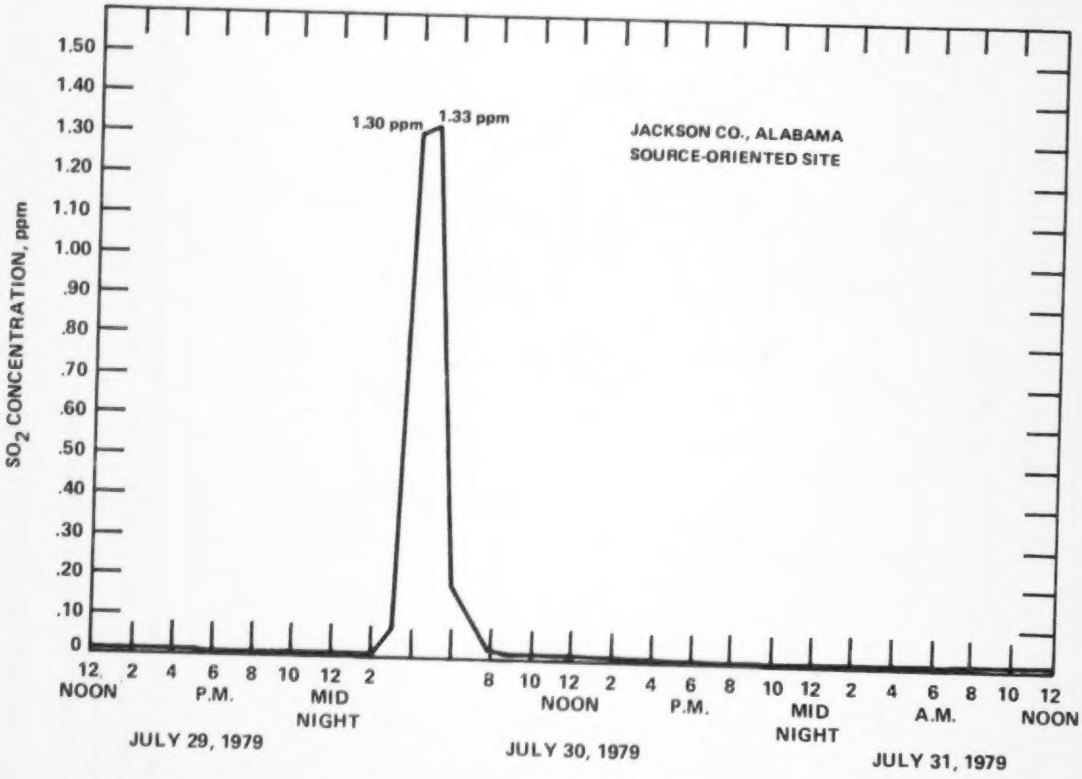

Figure 1-4. One example of rapid increase in ambient sulfur dioxide concentration from near zero to 1.30 ppm (3410 $\mu g/m^3$) during a period of approximately two hours.

Source: U.S. Environmental Protection Agency (1981).

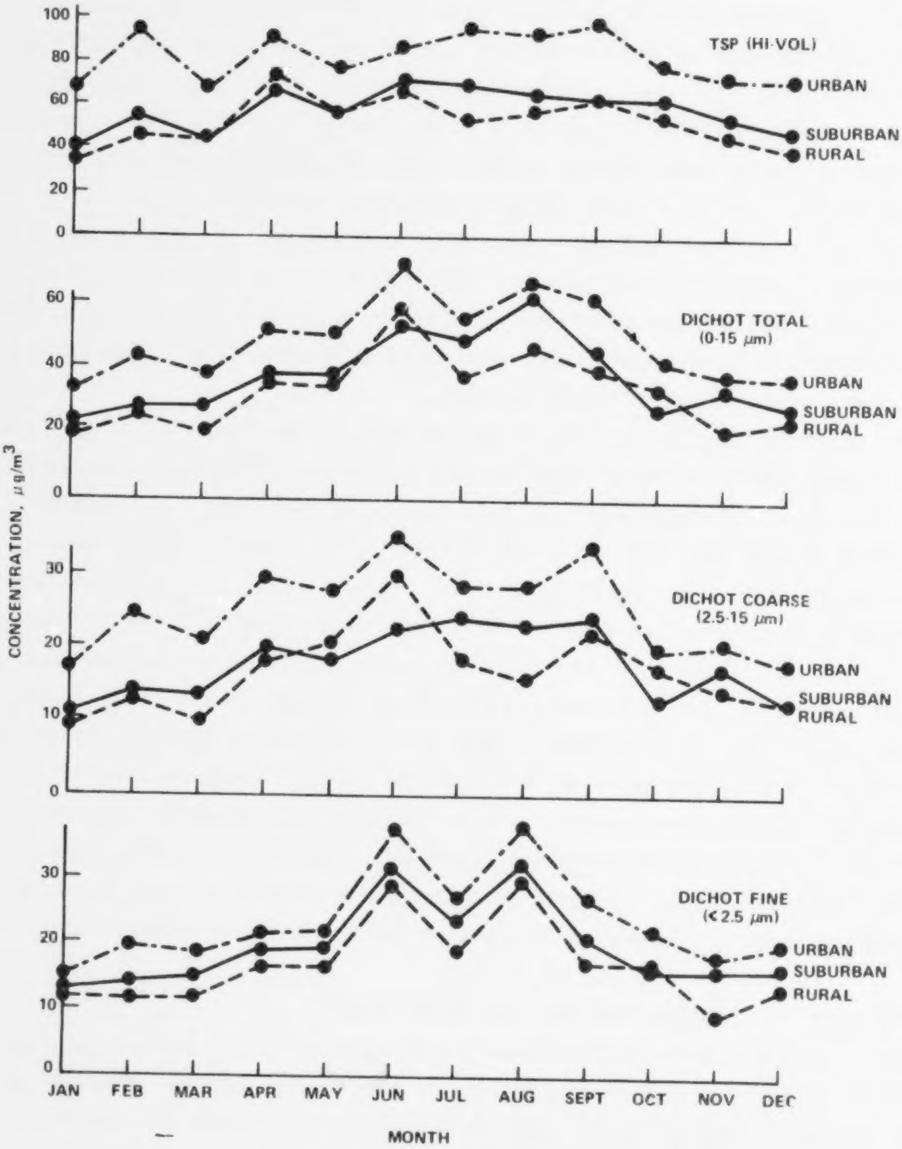

Figure 1-5. Seasonal variations in urban, suburban, and rural areas for four size ranges of particles. The data were obtained from a relatively small number of monitoring sites.

Source: After Trijonis et al. (1980).

Coarse particles are composed mainly of silica, calcium carbonate, clay minerals, and soot. Chemical constituents in the coarse fraction include silicon, aluminum, potassium, calcium, and iron, together with other alkaline-earth and transition elements. Organic materials are also found in coarse particles, including plant spores, pollens, and diverse biogenic detritus. Much of this coarse material is road dust suspended by traffic action. Street levels of resuspended dust can be very high. Traffic on unpaved roads generates huge amounts of dust that deposits on vegetation and can be resuspended by wind action. Rain and snow can reduce these emissions, but as one study suggests, salting of roads when precipitation occurs under freezing conditions may be a major source of winter TSP. Industrial fugitive emissions, particularly from unpaved access roads, construction activity, rock crushing, and cement manufacturing, can be a major category of coarse particles.

A number of calculational methods, generally categorized as source-apportionment or source-receptor models, are being used to trace particle levels to their sources. The results from chemical element balance calculations or factor analysis are available for several cities. Apportionments for these cities are presented in Chapter 5 as examples of results to be expected by future applications of these methods.

Ambient air monitors measure pollutant concentrations at fixed locations. Most individuals in our highly mobile society move through a variety of exposure levels that can be higher or lower than might be deduced solely from the values reported by a community's ambient air monitors. Most people spend a majority of their time indoors, where lower respiratory rates are associated with lower activity levels. Indoor levels of SO_2, which are almost entirely attributable to penetration from outdoors, can range from 10 to 90 percent of outdoor levels, depending on such factors as the tightness of house construction and the absorptive properties of walls, floors, and furniture. Presence or absence of air conditioning, rates of air exchange, and activities that resuspend dust influence indoor particulate matter levels. In addition, outdoor fine particles penetrate into buildings. Peak indoor TSP levels correlate to some degree with outdoor values, with a time lag that depends on a building's air-exchange rate. Because stationary ambient-air pollution monitors provide general statistics on composite population exposures, it would be extremely difficult (if not impossible) to predict an individual's actual exposure to SO_x and PM on the basis of community air-monitoring data alone.

1.6 ATMOSPHERIC TRANSPORT, TRANSFORMATION, AND DEPOSITION

The concentration of a pollutant at some fixed time and place beyond its source depends on: (1) the rate of emission and configuration of the source, (2) the chemical and physical reactions that transform one pollutant species to another, (3) the transport and diffusion (dilution) of the pollutant as a result of various meteorological variables, and (4) the removal of the pollutant through interaction with various surfaces on land and water (dry deposition) and interaction with rain drops or cloud particles (wet deposition). Figure 1-6 schematically illustrates some of these processes.

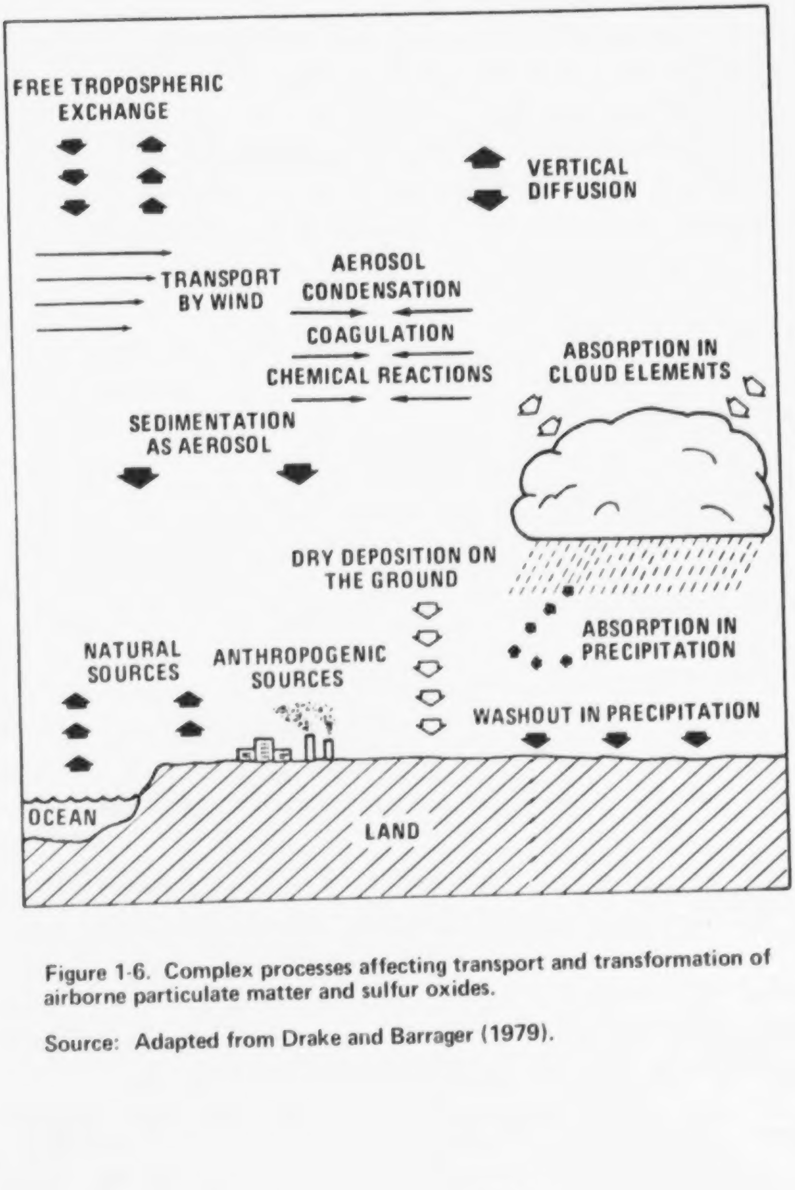
FREE TROPOSPHERIC EXCHANGE
VERTICAL DIFFUSION
TRANSPORT BY WIND
AEROSOL CONDENSATION
COAGULATION
CHEMICAL REACTIONS
ABSORPTION IN CLOUD ELEMENTS
SEDIMENTATION AS AEROSOL
DRY DEPOSITION ON THE GROUND
ABSORPTION IN PRECIPITATION
NATURAL SOURCES
ANTHROPOGENIC SOURCES
WASHOUT IN PRECIPITATION
OCEAN
LAND

Figure 1-6. Complex processes affecting transport and transformation of airborne particulate matter and sulfur oxides.

Source: Adapted from Drake and Barrager (1979).

Processes governing transport and diffusion, chemical transformation, and wet and dry removal of SO_2 and particulate matter are extremely complex and not completely understood. The oxidation rate of SO_2 observed in urban and rural atmospheres is only partially accounted for by gas-phase reactions. Liquid-phase catalytic reactions involving manganese, iron, and carbon may contribute to observed rates, but further research is required to determine the rates and detailed mechanisms of these processes under typical atmospheric conditions.

Dry deposition of SO_2 is fairly well understood as a result of extensive measurements over various surfaces. The study of particle deposition has focused on modeling physical aspects of the process, namely, aerodynamics. Few measurements for particles with compositions typical of those in polluted atmospheres exist to support these modeling efforts. Coarse particles are removed from the atmosphere much more rapidly than fine particles, for the residence time of fine particles in the atmosphere is apparently on the order of 1 week and their transport distance may exceed 500 km.

Understanding of wet-removal processes for SO_2 has progressed considerably in recent years, particularly in the area of solution-phase chemistry of rain drops. Removal of gases as well as particles depends mainly on the physical character of precipitation events, which in many instances may be the determining factor in how accurately wet-removal rates can be predicted.

Characterization of the dynamics of the planetary boundary layer is essential to an adequate understanding of pollutant transport and diffusion over all spatial scales. Though considerable advances have been made in this area, the ability to predict mean transport and diffusion over long distances is less than adequate, partly because of sparse spatial and temporal measurements of upper-air wind activity.

The long-range transport of the fine-particle/SO_2 complex results in the superposition and chemical interaction of emissions from many different types of sources. Present long-range air pollutant transport models are characterized by simple terms representing chemical transformation and wet and dry removal, and by varying degrees of sophistication in their treatment of transport and diffusion. None of the models adequately treats the dynamics of the planetary boundary layer. Although always limited by the adequacy of their underlying data bases, with further research and development long-range transport models should be able to address issues associated with the movement of pollutants over long distances.

1.7 ACIDIC DEPOSITION

The occurrence of acidic deposition, especially in the form of acidic precipitation (rain and snow), has become a matter of environmental concern. Acidic precipitation in various regions of the United States and elsewhere in the world has been associated with acidification of ponds, lakes, and streams, with a resultant disappearance of aquatic animal and plant life. Acidic precipitation is also believed to have the potential for leaching elements from sensitive soils and causing direct and indirect injury to forests and vegetation. It is also believed to play a role in damaging stone monuments and buildings and in corroding metals and deteriorating paint.

Chapter 7 of this document emphasizes the effects of the wet deposition of sulfur and nitrogen compounds on aquatic and terrestrial ecosystems. Dry deposition also plays an important role, but contributions by this process have not been well quantified. Because sulfur oxides and nitrogen oxides are closely linked in the formation of acidic precipitation, the present discussion is not limited to sulfur oxides. A critical assessment of the various causes as well as effects of acidic deposition will be presented in a future EPA document.

Acidic precipitation has been conventionally defined as precipitation with a pH less than 5.6, because precipitation formed in a geochemically clean environment would have a pH of approximately 5.6 due to the combining of carbon dioxide with water in the air to form carbonic acid. As shown in Figure 1-7, the acidity of precipitation in the Eastern United States currently averages from pH 3.9 to 5.0; and even in regions of the United States with average pH levels above 5.0, precipitation episodes with pH levels as low as 3.0 have been reported. Measurements have been weighted according to rainfall amounts in the calculation of the average values shown in Figure 1-7.

The pH level can vary during a precipitation event, from event to event, from season to season, and from geographical area to geographical area. Other substances in the atmosphere besides oxides of sulfur and nitrogen can also cause a shift in the pH of precipitation by making it more acidic or more basic. For example, dust and debris swept up from the ground by winds may become components of precipitation and affect its pH. In the West and Midwest, soil particles tend to be basic, but in the Eastern United States they tend to be acidic. Furthermore, in coastal areas sea spray strongly influences precipitation chemistry by contributing calcium, potassium, chlorine, and sulfates. In the final analysis, the pH of precipitation reflects the contributions of all of these components.

It is not known when precipitation in the United States began to become markedly acidic. Some scientists argue that it began with the burning of large amounts of coal in the industrial revolution, and others estimate that it began in the United States with the introduction in the 1950s of tall stacks on power plants. Still other scientists disagree completely and argue that rain has always been acidic. No definitive answer to the question exists at the present time. Because the pH of rain has not been monitored without interruption over extended periods of time, there are insufficient data to characterize with confidence long-term temporal trends in the pH of precipitation in the United States.

Though wet deposition is usually emphasized, it is not the only process by which acids or acidifying substances are added to bodies of water or to the land. Dry deposition also occurs. Dry-deposition processes include gravitational settling of particles, impaction of aerosols, and absorption of gases by soil or water or by objects at the earth's surface. Dew, fog, and frost are also involved in deposition processes but do not strictly fall into the category of wet or dry deposition. Dry deposition of particulate matter is not as well understood as wet deposition; however, it is known that both deposition processes contribute to

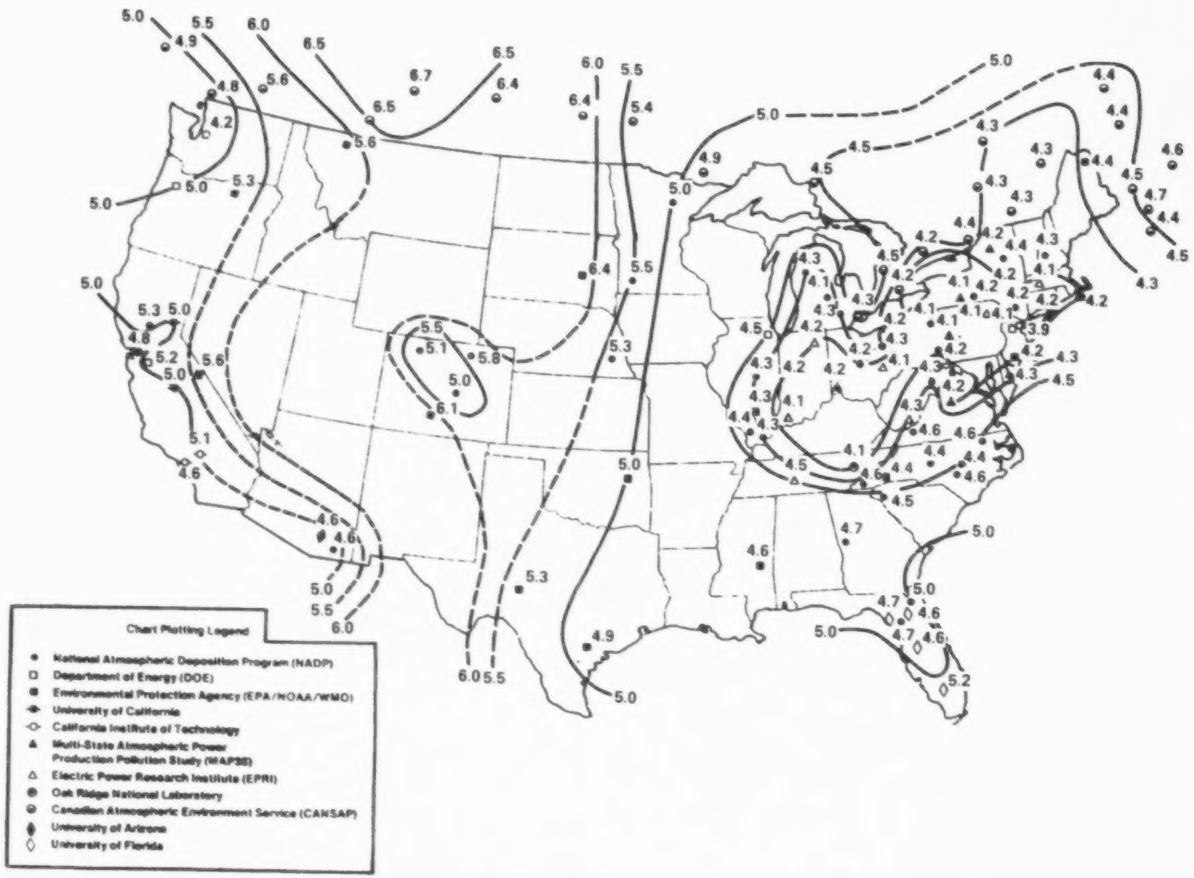
Chart Plotting Legend
• National Atmospheric Deposition Program (NADP)
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■ Environmental Protection Agency (EPA/NOAA/WMO)
◆ University of California
◇ California Institute of Technology
▲ Multi-State Atmospheric Power Production Pollution Study (MAP3S)
△ Electric Power Research Institute (EPRI)
● Oak Ridge National Laboratory
● Canadian Atmospheric Environment Service (CANSAP)
♦ University of Arizona
◇ University of Florida
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Figure 1-7. Average pH isopleths as determined from laboratory analyses of precipitation samples, weighted by the reported quantity of precipitation.

Source: Wisniewski and Keitz (1981).

the gradual accumulation of acidic or acidifying substances in the environment. Therefore, the reported effects of acidic deposition should not be attributed to wet deposition alone.

The most notable changes associated with acidic deposition are those observed in lakes and streams in New York's Adirondack Mountains, in Maine, in northern Florida, in the Precambrian Shield areas of Canada, in Scotland, and in the Scandinavian countries. In these regions, the decrease in the pH of freshwater bodies has been associated with changes in aquatic animal and plant populations. The chemistry of freshwaters is determined primarily by the geological structure (soil system and bedrock) of the lake or stream catchment basin, by the ground cover and by land use. In coastal regions marine salts also may be important in determining the chemical composition of freshwater streams, rivers, and lakes. The capability of a lake and its drainage basin to neutralize incoming acidic substances, however, is determined largely by the composition of the bedrock. Acidification of surface waters results when the sources of hydrogen ion exceed the ability of an ecosystem to neutralize the hydrogen ion. In general, the soils and crust of the earth are composed principally of basic materials with large capacities to buffer acids. However, areas where bedrock is particularly resistant to weathering and where soils are thin and poorly developed have much less neutralizing ability. This inability to neutralize hydrogen ions does not usually arise from a limited soil or mineral buffering capacity. Instead, low cation exchange capacity and slow mineral dissolution rates in relation to the relatively short retention time of water within the soil system may result in incomplete neutralization of soil waters and acidification of surface waters.

The capacity of organisms to withstand injury from weather extremes, pesticides, acidic deposition, or polluted air follows the ecological principle of limiting factors: For each physical factor in the environment there exists for each organism a minimum and a maximum limit beyond which no members of a particular species can survive. Either too much or too little of a factor such as heat, light, water, or minerals can jeopardize the survival of an individual and, in extreme cases, a species. The range of tolerance (see Figure 1-8) may be broad for one factor and narrow for another. The tolerance limit for each species is determined by its genetic makeup and therefore varies from species to species. The range of tolerance also varies depending on the age, stage of growth, and/or growth form of an organism. Limiting factors are, therefore, those components of an ecosystem which, when scarce or overabundant, limit the growth, reproduction, or distribution of an organism.

The stability of natural ecosystems under stress from marked environmental changes or perturbations depends upon the ability of the constituent organisms to adapt and to continue reproduction of their species. The most sensitive species decline or die out first. However, the capacity of an ecosystem to maintain internal stability is determined by the ability of all constituent organisms to adjust and survive. Thus, other species may be subsequently affected due to the loss of the most susceptible species.

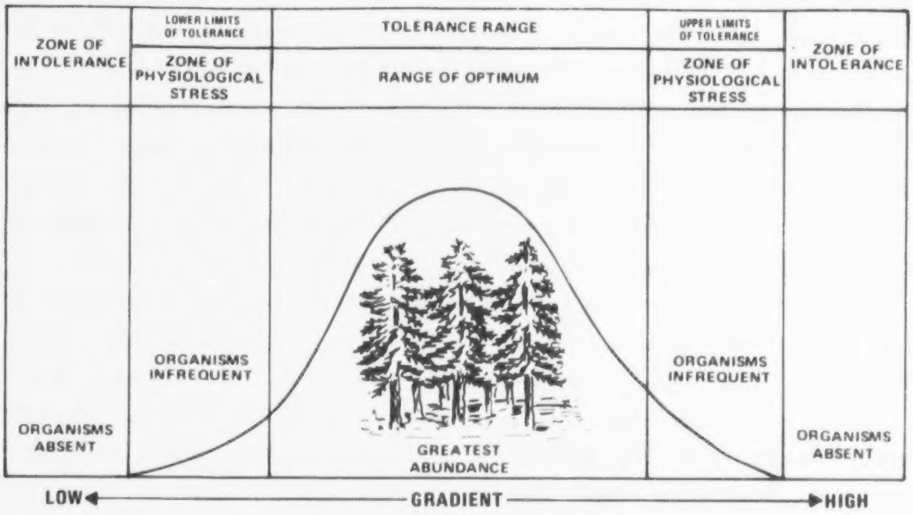

ZONE OF INTOLERANCE
LOWER LIMITS OF TOLERANCE
ZONE OF PHYSIOLOGICAL STRESS
TOLERANCE RANGE
RANGE OF OPTIMUM
UPPER LIMITS OF TOLERANCE
ZONE OF PHYSIOLOGICAL STRESS
ZONE OF INTOLERANCE
ORGANISMS ABSENT
ORGANISMS INFREQUENT
GREATEST ABUNDANCE
ORGANISMS INFREQUENT
ORGANISMS ABSENT
LOW
GRADIENT
HIGH

Figure 1-8. Idealized conceptual framework illustrating the "law of tolerance," which postulates a limited range of various environmental factors within which species can survive.

Source: Adapted from Smith (1980).

Continued or severe disturbance of an ecosystem can overcome its resistance or prevent its recovery, with the result that it is replaced by a new system. In the Adirondack Mountains of New York State, in eastern Canada, and in parts of Scandinavia the original aquatic ecosystems have been and are continuing to be replaced by ecosystems different from the original because of acidification of the aquatic habitat. Forest ecosystems, however, appear thus far to have been resistant to changes due to perturbation or stress from acidifying substances.

The disappearance of fish populations from freshwater lakes and streams is usually one of the most readily observable signs of lake acidification. The death of fish in acidified waters has been attributed to the modification of a number of physiological processes. The sensitivity of fish to low pH levels has also been shown to depend on aqueous calcium levels. The reproductive failure of fish has been cited as the primary factor leading to the gradual extinction of fish populations. Long-term gradual increases in acidity, particularly below pH 5, interfere with reproduction and spawning, producing a decrease in population density and a shift in size and age characteristics of the population toward larger and older fish. Such effects often are not recognizable until the population is close to extinction, particularly in the case of late-maturing species with long lives. Even relatively small increases (as low as 5 percent) in mortality of fish eggs and fry can decrease reproduction and bring about extinction.

Acidic pollutants deposited during the winter accumulate in the snowpack and ice, and may be released in a relatively short time during the melting of the snowpack and ice cover in the spring. The resulting sudden short-term changes in water chemistry may have a significant impact on aquatic biota, especially if they occur during spawning or during early stages of development or other points in the life cycle when the organisms are particularly vulnerable.

In some acidified lakes, concentrations of aluminum may be equally and perhaps more important than pH levels as a factor causing a decline in fish populations. At low pH levels certain aluminum compounds in the water may be mobilized, thereby upsetting the osmoregulatory function of the blood in fish. Aluminum toxicity to aquatic organisms other than fish has not been assessed.

When evaluating the potential effects of acidification on fish or other biotic populations, it is very important to keep in mind the highly diversified nature of aquatic systems spatially, seasonally, and year-to-year. As a result of this diversity, it is necessary to evaluate each system independently in assessing the reaction of a population to acidification. Survival of a fish population may depend more on the availability of refuge areas from acid conditions during spring melt than on mean annual pH, calcium, or inorganic aluminum levels.

Other organisms ranging from bacteria to waterfowl may also be affected by lake and stream acidification. Organisms at all levels in the food web appear to be vulnerable. Reductions in the number and diversity of species may occur, biomass (total mass of living organisms in a given volume of water) may be altered, and processes such as primary production and decomposition may be impaired.

Significant changes that have occurred in aquatic ecosystems with increasing acidity, particularly as the pH drops below 5.5, include the following:

1. Fish populations are reduced or eliminated.
2. Bacterial decomposition is reduced, and fungi may become dominant in aquatic communities that feed on organic debris. Consequently, such matter accumulates rapidly, tying up nutrients and limiting nutrient mineralization and cycling.
3. Species diversity and total numbers of species of aquatic plants and animals are reduced. Acid-tolerant species predominate.
4. Phytoplankton productivity may be reduced because of changes in nutrient cycling and nutrient limitations.
5. Biomass and total productivity of benthic macroscopic plants and algae may increase, in part because of increased lake transparency.
6. The number and biomass of herbivorous invertebrates decline. Tolerant invertebrate species, such as air-breathing insects, may become abundant primarily because of reduced fish predation.
7. Changes in community structure occur at all levels in the food web.

An indirect effect of acidification potentially of concern to human health is the possible contamination of edible fish and of water supplies. Studies in Canada and Sweden reveal high concentrations of mercury in fish from acidified regions. Potentially toxic levels of lead have been found in plumbing systems with acidified water, and persons drinking the water could be affected by the lead. However, no cases have yet been documented of human health effects being directly linked to the impact of acidic precipitation on water supplies or edible aquatic organisms.

Soils may become gradually acidified from an influx of hydrogen ions. Leaching of the mobilizable forms of mineral nutrients may occur. The rate of leaching is determined by the buffering capacity of the soil and the amount and composition of precipitation. Anion mobility is also an important factor in the leaching of soil nutrients, for cations cannot leach without the associated anions also leaching. The capacity of soils to adsorb and retain anions increases when hydrated oxides of iron and of aluminum are present.

Sulfur and nitrogen are essential for optimal plant growth. Plants usually obtain sulfur in the form of sulfate from organic matter during microbial decomposition. Wet and dry deposition of atmospheric sulfur is also a major source. In soils where sulfur and nitrogen are limiting nutrients, such deposition may increase growth in some plant species. The amount of sulfur entering the soil system from the atmosphere depends on proximity to industrial areas, sea coast, and marshlands. The prevailing winds and the amount of precipitation in a given region are also important. Near fossil-fuel power plants and industrial installations the amount of sulfur in precipitation may be as much as 168 kilograms per hectare (150 pounds per acre) or more.

At present there are no documented observations or measurements of changes either in natural terrestrial ecosystems or in agricultural productivity directly attributable to acidic precipitation under ambient conditions. Information regarding effects on vegetation comes from controlled research studies, which mainly use some form of simulated acidic rain such as dilute sulfuric acid. These simulated rains have deposited hydrogen (H^+), sulfate (SO_4^{2-}), and nitrate (NO_3^-) ions on vegetation and caused necrotic lesions in a wide variety of plant species under greenhouse and laboratory conditions. Such results must be interpreted with caution, however, because the growth and morphology of leaves under such conditions are not necessarily typical of field conditions. Studies of the effects of simulated acidic precipitation on field crops have reported beneficial, detrimental, or no effects on yield, depending on the particular species as well as the portion of the plant that is of economic value (e.g., root, leaf, fruit).

Damage to monuments and buildings made of stone, corrosion of metals, and deterioration of paint may be caused by acidic precipitation, but the effects resulting from dry or wet deposition of sulfur compounds cannot be clearly distinguished. Also, deposition of sulfur compounds on stone surfaces may cause damage indirectly by providing a medium for microbial growth that can result in deterioration.

Several aspects of the phenomenon of acidic precipitation remain subject to debate because of ambiguous or inadequate data. Important unresolved issues include:

(1) the rate at which rainfall is becoming more acidic and/or the rate at which the phenomenon is becoming geographically more widespread;

(2) the relative extent to which the acidity of rainfall in a region depends on local emissions of nitrogen and sulfur oxides versus emissions transported from distant sources;

(3) the relative importance of changes in total mass-emission rates compared to changes in the nature of the emission patterns (e.g., ground-level versus tall-stack emissions) in contributing to the regional acidification of precipitation;

(4) the relative contribution of wet and dry deposition to the acidification of lakes and streams;

(5) the geographic distribution of natural sources of SO_x, nitrogen oxides (NO_x), and ammonia, and the significance of their seasonal as well as annual contributions;

(6) the existence and significance of anthropogenic, non-combustion sources of SO_x, NO_x, and hydrogen chloride (HCl);

(7) the dry deposition rates for SO_2, NO_x, sulfate, nitrate, and HCl over various terrains and at different seasons of the year;

(8) the existence and reliability of long-term pH measurements of lakes and headwater streams;

(9) the acceptability of current models for predicting long-range SO_x and NO_x transport and of models for predicting the acid-tolerance of lakes;

(10) the feasibility and costs of using liming or other corrective procedures to prevent or reverse damage from acidification;

(11) the differential effects of sulfate, nitrate, and hydrogen ion deposition on ecosystem dynamics in both aquatic and terrestrial ecosystems;

(12) the effectiveness of fertilization resulting from sulfate and nitrate deposition on soils;

(13) the ultimate effects of acidic deposition on agricultural crops, forests, and other native plants; and

(14) the effects of acidic deposition on soil microbial processes and nutrient cycling.

A comprehensive critical assessment of scientific evidence bearing on these and other issues will be presented in a future EPA document on the causes and effects of acidic deposition.

1.8 EFFECTS ON VEGETATION

Plants may be exposed to sulfur dioxide and particulate sulfate through dry and wet deposition. Of the two, sulfur dioxide is potentially more injurious to vegetation, particularly when it is in combination with other airborne pollutants. The effects of SO_2 through external exposure of vegetation or through contact with the soil substrate are much more difficult to assess than the effects associated with the entry of SO_2 into the plant.

To cause injury, sulfur dioxide must enter a plant through leaf openings, or stomata. After entering plant cells through the stomata, sulfur dioxide is converted to sulfite and bisulfite, which may then be oxidized to sulfate. Sulfate is about 30 times less toxic than sulfite and bisulfite. Absorption rates and plant resistance to sulfur have been shown to vary with different species exposed to SO_2. For example, sulfur dioxide has been shown to induce stomatal closure in some plants and to induce stomatal opening in others. In some instances, tolerance to SO_2 may depend less on the amount of pollutant absorbed than on the ability of the plant to move SO_2 out of the leaf and into other plant tissues. As long as the absorption rate of SO_2 in plants does not exceed the rate of conversion to sulfate, the only effects of exposure may be changes in opening or closing of stomata, or subtle changes in the biochemical or physiological systems. Such effects may abate if SO_2 concentrations are reduced. Pollutant uptake by plants may be influenced by such dynamic physical factors as light, leaf surface moisture, relative humidity, and soil moisture. Such factors influence internal physiological conditions in plants as well as stomatal opening and closing and, therefore, play a major role in determining the sensitivity of the plant species or cultivars.

Symptoms of SO_2-induced injury in higher plants may be quite variable, since response is governed by pollutant dose (concentration multiplied by duration of exposure), conditions of exposure (e.g., day vs. night, peak vs. long-term), physiological status of the plant, maturational stage of plant growth, environmental influences on the pollutant/plant interaction, and environmental influences on the metabolic status of the plant itself. Although the product of time and concentration may remain constant, the effect of exposure may vary for a given dose. The relationship between exposure and injury is generally more influenced by changes in concentration than by changes in duration of exposure.

Possible plant responses to SO_2 and related sulfur compounds include: (1) increased growth and yield due to fertilization effects; (2) no detectable response; (3) injury manifested as growth and yield reductions without visible symptoms on the foliage or with very mild foliar symptoms that would be difficult to attribute to air pollution without comparing control plants grown under pollution-free conditions; (4) injury exhibited as chronic or acute symptoms on foliage with or without associated reduction in growth and yield; and (5) death of plants or plant communities.

Under certain conditions, atmospheric SO_2 can have beneficial effects on agronomic vegetation. The amount of sulfur accumulated from the atmosphere by leaf tissues is influenced by the amount of sulfur in soil relative to the sulfur requirement of the plant. After exposure to low doses of SO_2, plants grown in sulfur-deficient soils have exhibited increased productivity.

As the concentration of SO_2 increases, plants may develop more predictable and more obvious visible symptoms. Foliar symptoms progress from chlorosis, or other types of pigmentation changes, to the development of necrotic areas, the extent of which increases with exposure. Studies of the effects of SO_2 on growth and yield have demonstrated a reduction in the dry weight of foliage, shoots, roots, and seeds, as well as a reduction in the number of seeds. At still higher doses there are further reductions in growth and yield. Extensive mortality has been noted in forests continuously exposed to SO_2 for many years. The presence of acute or chronic foliar injury does not necessarily indicate that growth or yield is affected, nor does foliar injury always portend subsequent growth or yield effects. However, plant productivity and visible damage to foliage are the best available indicators of plant response to SO_2.

A number of species of plants, particularly lichens, are sensitive to low concentrations of SO_2, and some may be used as bioindicators of such pollution. Even sensitive species may be asymptomatic, however, depending on the environmental conditions before, during, and after exposure to SO_2. Because of the absence of empirical data quantifying losses in growth or yield in relation to SO_2 exposure, sensitive species are generally identified on the basis of visible symptoms.

Dose-response studies aid in quantifying plant response to air pollutants. Useful generalizations on the relationship between parameters of plant response and measurable indices of dose may be developed. "Response" may be considered to be a measurable change in parameters such as gas-exchange rates, photosynthetic rates, biochemical pathways, physiological functions, degree of visible leaf injury, and subsequent effects on growth and yield. In interpreting dose-response studies, it is important to realize that the ultimate effect of a given exposure dose may be influenced significantly by environmental factors that control the rate of pollutant flux into plant leaves and by plant factors that determine the metabolic fate of the pollutant within leaf tissues (see Figure 1-9).

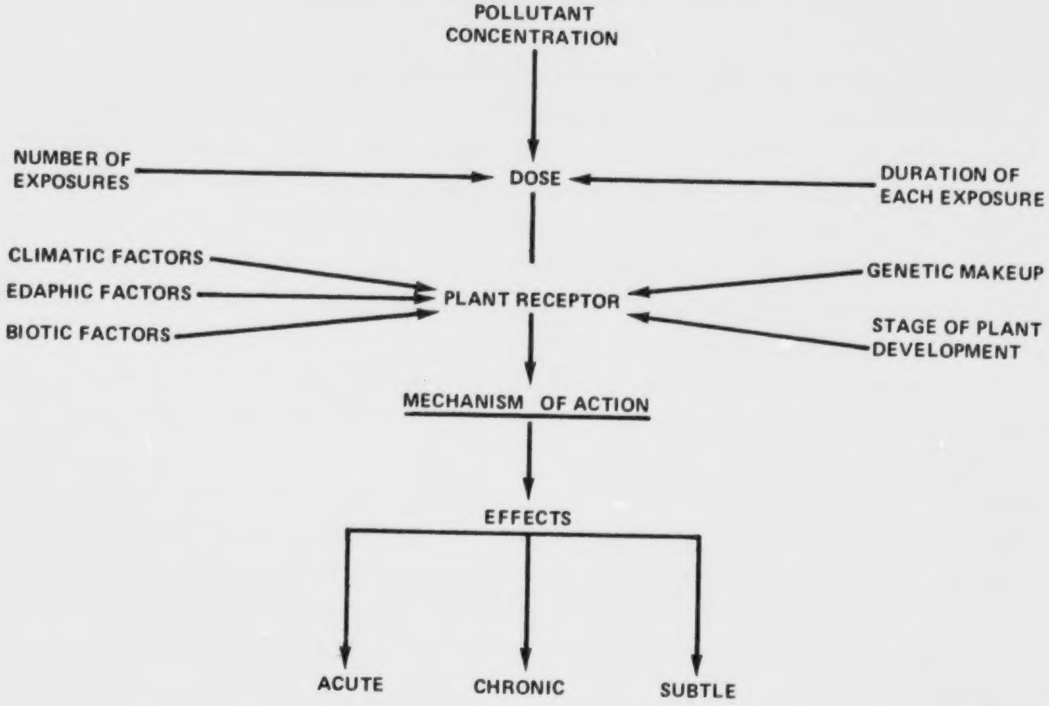

POLLUTANT CONCENTRATION
NUMBER OF EXPOSURES
DOSE
DURATION OF EACH EXPOSURE
CLIMATIC FACTORS
EDAPHIC FACTORS
BIOTIC FACTORS
PLANT RECEPTOR
GENETIC MAKEUP
STAGE OF PLANT DEVELOPMENT
MECHANISM OF ACTION
EFFECTS
ACUTE
CHRONIC
SUBTLE

Figure 1-9. Conceptual model of the factors involved in air pollution's effects on vegetation.

Source: Heck and Brandt (1977).

Concentrations of SO_2 from point sources may fluctuate widely within short periods. Laboratory experiments have demonstrated that short-term exposures at high concentrations of SO_2 are relatively more toxic than longer-term exposures with the same total dose. In studies of the effects on kidney beans of short-term (3-hr) SO_2 exposures, increasing the peak-to-mean ratio of SO_2 concentrations from 1.0 to 2.0 did not alter the degree of depression in photosynthetic activity; however, increasing the peak-to-mean ratio to 6.0 tripled the depression in post-fumigation photosynthetic activity, although the total dose delivered remained approximately the same (i.e., in the 1.0 to 2.0 ppm-hr range).

Another important aspect of exposure dose is the frequency and duration of periods of low SO_2 stress. Such periods may be critical to the recovery of plant systems following exposure to elevated levels of SO_2. Thus, experimental studies using continuous exposure systems probably overestimate the toxicity of the delivered dose in many cases. Recovery would be more likely to occur under field conditions, where fluctuating meteorological conditions strongly influence exposure patterns.

Plant growth and development represent an integration of cellular and biochemical processes. The response of a given species or variety of plants to a specific air pollutant cannot be precisely predicted on the basis of the known response of related plants to the same pollutant; neither can the response of a plant be predicted on the basis of its response to similar doses of other pollutants. Each plant species is different genetically, and therefore its genetic susceptibility and the influence of the environment at the time of exposure must be considered for each plant and each pollutant. Because of the variation in response shown by different plant species and different cultivars of the same species, making generalizations is difficult. For example, studies (Dreisinger and McGovern, 1970) of SO_2 effects on vegetation in non-arid regions (where environmental conditions such as high temperature, high humidity, and abundant sunlight enhance plants' responsiveness to SO_2) indicate that many species of sensitive and intermediately responsive vegetation would likely, from time to time, show visible injury when exposed to peak (5-min), 1-hour, and 3-hour SO_2 concentrations as low as 2.6-5.2, 0.13-5.2, and 0.78-2.1 mg/m^3 (1-2, 0.5-2, and 0.3-0.8 ppm) respectively. In contrast, other studies (Hill et al., 1974) indicate that some species of vegetation in arid regions would probably not show visible signs of injury even at SO_2 concentrations as high as 28.82 mg/m^3 (11 ppm) for 2 hours.

In general, studies discussed in Chapter 8 indicate that regardless of the conditions of exposure, for a given plant species or variety there is a critical SO_2 concentration and duration of exposure above which plant injury will occur. Such injury results from exceeding the plant's capability to transform toxic SO_2 and sulfite into much less toxic sulfate and ultimately to transfer or break down the sulfate.

At present, data concerning the interactions of SO_2 with other pollutants indicate that, on a regional scale, SO_2 occurs at least intermittently at concentrations high enough to produce significant interactions with other pollutants, principally ozone. A major weakness in the understanding of pollutant interactions, however, is the lack of in-depth analysis of existing regional air-quality data sets for the three principal pollutants (SO_2, ozone, and nitrogen dioxide). Analysis of these data should show how frequently and at what concentrations the pollutants occur together both spatially and temporally within regions of major concern. The relative impact on plants of the simultaneous versus sequential exposure to these pollutants is also not well documented and is crucial in evaluating the likelihood and extent of potential pollutant interactive effects under field conditions.

A few studies have reported that combinations of particulate matter and SO_2, or particulate matter and other pollutants, may increase foliar uptake of SO_2, increase foliar injury of vegetation by heavy metals, and/or reduce growth and yield. Because of the complex nature of particulate pollutants, conventional methods for assessing pollutant injury to vegetation, such as dose-response relationships, are poorly developed. Studies have generally reported responses relative to a given source and the physical size or chemical composition of the particles. For the most part, studies have not focused on effects associated with specific ambient concentrations. Coarse particles, such as dust, directly deposited on leaf surfaces can reduce gas exchange, increase leaf surface temperature, reduce photosynthesis, and lead to chlorosis, reduced growth, and leaf necrosis. Heavy metals deposited either on leaf surfaces or on the soil and subsequently taken up by a plant can accumulate and reach toxic concentrations within the tissues of the plant.

Natural ecosystems are integral to the maintenance of the biosphere, and disturbances of these ecosystems may have long-range effects that are difficult to predict. In the United States anthropogenic contributions to atmospheric sulfur exceed natural sources; most of these emissions are deposited (by wet and dry deposition) on terrestrial and aquatic ecosystems. The subsequent fate and distribution of sulfur in these systems is not well understood. The wet deposition of sulfur compounds is discussed in Chapter 7.

Natural ecosystems do not respond to environmental perturbations in the same manner as do a few isolated individuals or crop monocultures. The responses observed in ecosystems under ambient conditions are a complex function of many variables, which cannot conclusively be attributed to any particular substance such as sulfur dioxide or particulate matter alone. Data relating responses of ecosystems to specific doses of SO_2 and other pollutants are difficult to obtain and interpret because of the generally longer periods of time over which these responses occur and because of the many biotic and abiotic factors that modify them.

Vegetation within terrestrial ecosystems is sensitive to SO_2 toxicity, as evidenced by changes in physiology, growth, development, survival, reproductive potential, and community composition. Indirect effects may result from the modification of the habitat through change in the decomposition of litter and the cycling of nutrients or through altered structure of the community. At the community level chronic exposure to SO_2, particularly in combination with other pollutants such as ozone may cause shifts in community composition as evidenced by elimination of individuals or populations sensitive to the pollutant. Differential effects on individual species within a community can occur through direct effects on sensitive species and through alteration of the relative competitive potential of species within the plant community. In one study of a forest chronically exposed to gaseous sulfur emissions, changes were observed in the mineral nutrient balance and in the biological relationships among the various components of the ecosystem. However, a reduction in gaseous sulfur emissions occurred during the study, and it appeared that the changes resulting from the ecological perturbations were reversible.

Particulate emissions have their greatest impact on terrestrial ecosystems near large sources of emission. Particulate matter in itself constitutes a problem only in those few areas where rates of deposition are very high. However, ecological modification may occur if the particles contain toxic elements, even though deposition rates are moderate. Solubility of particle constituents is a critical factor determining the impact of particulate matter deposited on terrestrial ecosystems since water-insoluble elements are not mobile within ecosystems. Most of the material deposited by wet and dry deposition on foliar surfaces in vegetated areas is transferred to the soil where accumulation in the litter layer occurs. Such accumulation may affect recycling of the nutrients in litter.

1.9 EFFECTS ON VISIBILITY AND CLIMATE

"Atmospheric visibility" is a term often used by airport weather observers to connote visual range, which refers to the farthest distance at which a large, black object can be seen against the horizon sky in the daytime. In the everyday sense, visibility relates to atmospheric clarity and the perceived characteristics of viewed surroundings, including the contrast and the color of objects and sky. Pollution affects visibility in two primary ways: (1) as coherent plumes or haze layers visible because of their contrast with background; and (2) as widespread, relatively homogeneous haze that reduces contrast of viewed targets and reduces visual range. The kind and degree of effects are determined largely by the distribution and characteristics of atmospheric particulate matter, which scatters and absorbs light.

The currently available methods for monitoring visibility measure different aspects of visibility impairment. Generally, contrast-type measurements (such as photography, telephotometry, and human eye observations) relate well to the perception of visual air quality, while extinction or scattering measurements (such as transmissometry and nephelometry) relate to the cause of visibility degradation. Each of these measurement methods can be used to approximate visual range. No single method is yet widely accepted for measuring light absorption.

Visibility in the United States, as indicated by airport observations, is depicted in Figure 1-10. Some uncertainty arises from the use of airport data to characterize regional visibility because of differences in target quality and observers between sites and at the same site. Despite these limitations, the data should be at least indicative of regional trends. The best visibility occurs in the mountainous Southwest where annual median visibility exceeds 110 km (70 miles). East of the Mississippi and south of the Great Lakes, annual median visibilities are less than 24 km (15 miles) and are significantly lower in summer, particularly during sporadic episodes of regional haze. To some extent, these regional differences reflect naturally occurring meteorological patterns, such as the higher humidity of the Southeast versus the Southwest.

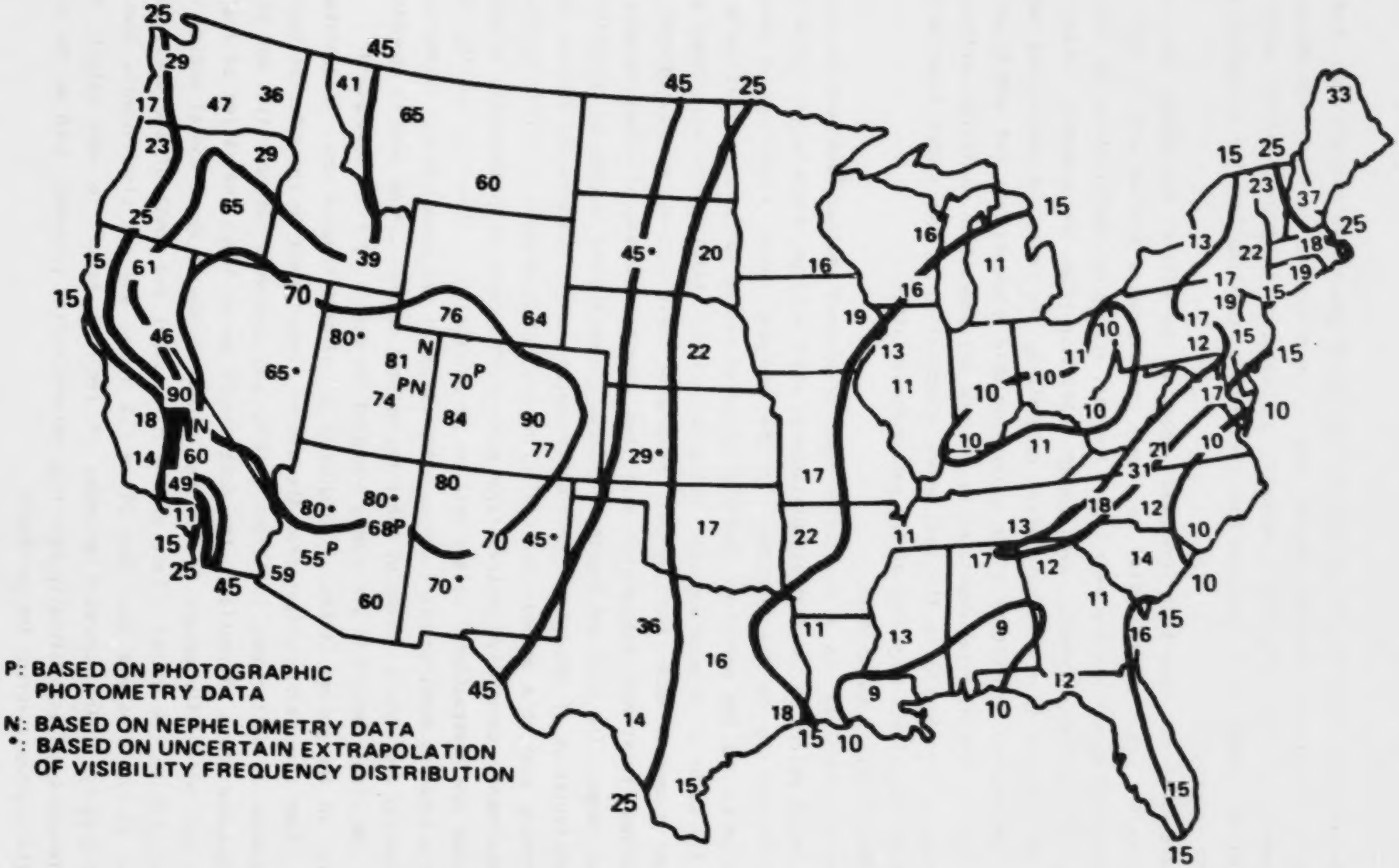

P: BASED ON PHOTOGRAPHIC PHOTOMETRY DATA
N: BASED ON NEPHELOMETRY DATA
•: BASED ON UNCERTAIN EXTRAPOLATION OF VISIBILITY FREQUENCY DISTRIBUTION

Figure 1-10. Median yearly visual range (miles) and isopleths for suburban/nonurban areas, 1974-1976. (1 mile = 1.6 km)

Source: Trijonis and Shapland (1979).

Visual range is inversely related to total extinction and can be estimated, if extinction is known, by the Koschmieder relationship (see Figure 1-11). Total extinction is the sum of scattering and absorption by particles and by gases. Because extinction is dependent on wavelength and sun angle, particle-derived haze may appear blue, white, gray, or brown under varying conditions. On a regional scale, visibility reduction is generally dominated by light scattering by fine particles, particularly those in the 0.1 to 2 μm size range. In urban areas, absorption of light by fine carbonaceous particles (and, to a lesser extent, NO_2) can become important.

Extinction due to scattering is closely proportional to the fine-particle mass concentration (Figure 1-12), with typical extinction/fine mass concentration ratios (for <70-percent relative humidity) of about 3 m^2/g. Measurements suggest that extinction due to fine-particle scattering will increase by a factor of two to three as relative humidity (RH) is increased from 50 to 90 percent. This increase is due to absorption of atmospheric water vapor by aerosol constituents such as sulfates. Despite the well-established functional relationship between visual range and fine-particle mass concentration, the choice of fixed coefficients for the relationship is complicated by the spatial and temporal variation of RH, particle composition, and observer contrast thresholds.

The major constituents of fine particles from natural and anthropogenic sources contribute in varying degrees to the impairment of visibility. Theoretical and empirical findings suggest that two constituents, sulfates and elemental carbon, generally tend to be most significant. Sulfate, with associated ammonium and water, often dominates the fine-particle mass and hence impairment of visibility. In urban areas, elemental carbon can be a major visibility-reducing species. In both cases, significant variations can occur at different times and sites. Other species, such as nitrates and organics, may also be important, but understanding of their roles is hindered by lack of reliable data.

Studies of trends in airport visibility in the Eastern United States indicate that, while wintertime visibilities improved in some northeastern locations, visibility in the East declined overall (Figure 1-13). Summer, often the season of best visibility in the early 1950s, is currently the season of worst visibility. From 1948 to 1974, summertime haze (extinction) increased by more than 100 percent in the Central Eastern States, by 50 to 70 percent in the Midwest and the Eastern Sunbelt States, and by 10 to 20 percent in the New England area. Although the results of airport surveys should be viewed with caution, the results are consistent from site to site. Similarities exist in the long-term record of the spatial and seasonal trends in airport visibility, sulfate concentrations, and point-source emissions of SO_x. These similarities suggest, but do not prove, that historical visibility trends in the East were caused, at least in part, by regional sulfur oxides emissions and resultant sulfate aerosol concentrations.

Reductions in visibility can adversely affect transportation safety, property values, and aesthetics. When visibility (visual range) drops below 4.8 km (3 miles), Federal Aviation

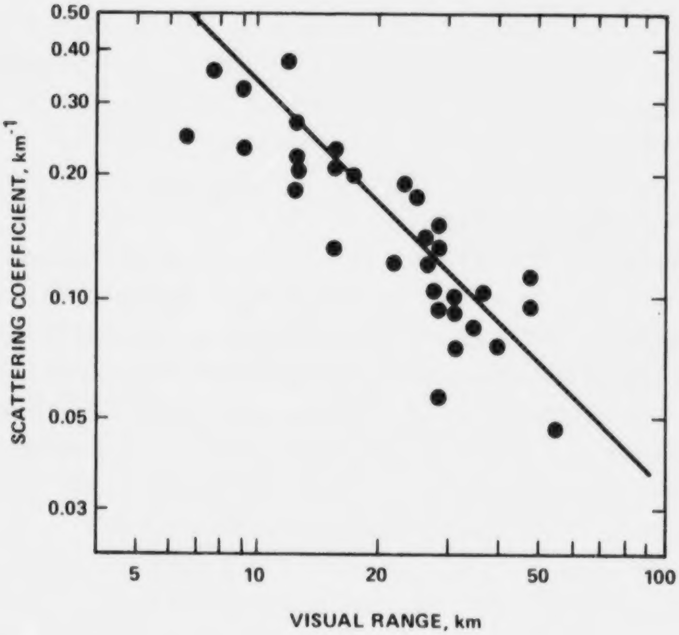


Figure 1-11. Inverse proportionality between visual range (V) and the scattering coefficient (σ_{sp}) as measured at the point of observation. The straight line is derived from the Koschmieder formula for visual range, assuming $V = 3.9/\sigma_{sp}$ and nonabsorbing media ($\sigma_{ext} = \sigma_{sp}$). The correlation coefficient for V and σ_{sp} is −0.89.

Source: Horvath and Noll (1969).

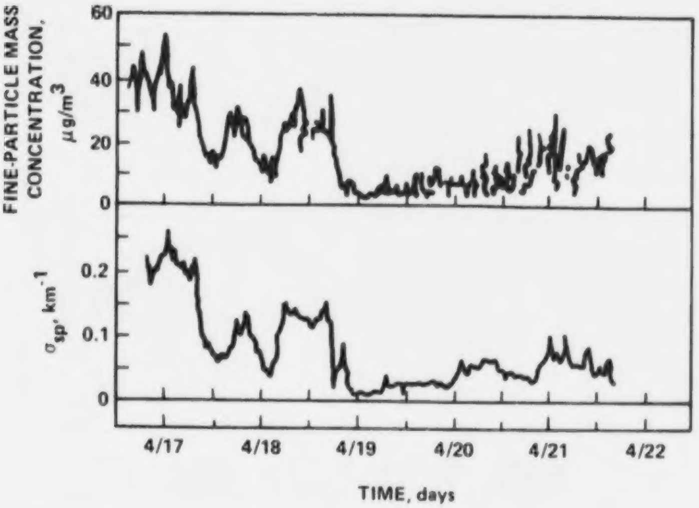
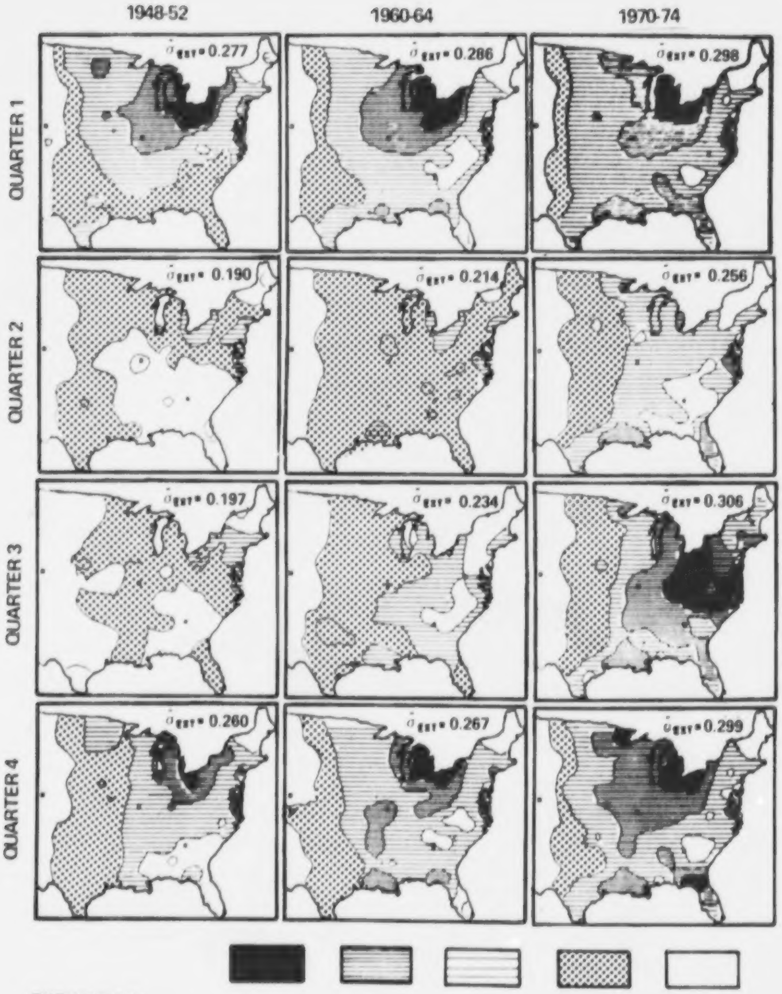


Figure 1-12. Simultaneous in situ monitoring of σ_{sp} and fine-particle mass concentration in St. Louis in April 1973 showed a high correlation coefficient of 0.96, indicating that σ_{sp} depends primarily on the fine-particle concentration.

Source: Macias and Husar (1976).



1948-52
1960-64
1970-74
QUARTER 1
QUARTER 2
QUARTER 3
QUARTER 4
σEXT = 0.277
σEXT = 0.286
σEXT = 0.298
σEXT = 0.190
σEXT = 0.214
σEXT = 0.256
σEXT = 0.197
σEXT = 0.234
σEXT = 0.306
σEXT = 0.260
σEXT = 0.267
σEXT = 0.299

	■	▤	☰	▨	□
EXTINCTION COEFFICIENT, km^{-1}	>0.36	0.3-0.36	0.24-0.30	0.18-0.24	<0.18
VISIBILITY, miles	<6.6	6.6-8	8-10	10-13.3	>13.3
(km)	(< 11)	(11-13)	(13-16)	(16-21)	(>21)

Figure 1-13. The spatial distribution of 5-year average extinction coefficients shows the substantial increases of third-quarter extinction coefficients in the Carolinas, Ohio River Valley, and Tennessee-Kentucky area. In the summers of 1948-1952, a 1000-km size multistate region around Atlanta, GA, had visibility greater than 24 km (15 miles); visibility had declined to less than 13 km (8 miles) by the 1970s. The spatial trend of winter (first quarter) visibility shows improvements in the Northeast megalopolis region and some worsening in the Sunbelt region. Both spring and fall quarters exhibit moderate but detectable increases over the entire Eastern United States.

Source: Husar et al. (1979).

Administration regulations restrict flight in controlled air spaces to those aircraft equipped with instrument flight rules (IFR) instrumentation. Under these conditions, most small aircraft would be grounded. In addition, for some airports operating under IFR conditions during periods of peak traffic, delays in arrivals and departures might occur. Airport visibility data from the National Weather Service indicate that during the summer months about half of all visibilities less than 4.8 km (3 miles) at noontime occur in the absence of fog, precipitation, or blowing material. Preliminary studies of the economic value of visibility, conducted in both urban and nonurban settings, show that the public is concerned and willing to pay for improved visual air quality. These studies are still too limited, however, to permit any large-scale quantitative evaluation.

The relatively long residence time and light-attenuating properties of fine particles may lead to slow and subtle changes in the nature of the atmosphere and, possibly, in climate. Three possible effects have been recognized, each of which may have far-reaching and interrelated consequences. First, the amount of solar energy reaching ground level may be reduced, some being backscattered to space and some being diverted to increased atmospheric heating. Thus, less energy will be available at ground level for photosynthesis and commercial exploitation of solar energy. Second, reductions in solar radiation may lead to alterations in local or regional temperatures, which may lead to changes in atmospheric stability, agricultural production, energy usage, and sea level. Third, increased cloud formation may alter precipitation patterns, which may lead to changes in agricultural production. The complex radiative interactions between atmosphere and earth have obscured the influence of fine particles on temperature balance and precipitation patterns. The role of fine particles in reducing ground level solar radiation is better understood.

If there are no clouds between the observer and the sun, the intensity of direct solar radiation for a given solar elevation depends on the variable amount of dust, haze, and water vapor in the atmosphere. The extinction produced by these constituents is called "atmospheric turbidity." Data from a 29-station turbidity network showed that there are strong spatial and temporal variations of turbidity across the United States. The mean annual net loss of ground level solar radiation due to particles for 1961-1966 was estimated at 2.6 percent for the Southwestern United States and 4 percent for the Midwestern states, based on reasonable assumptions for the amount backscattered to space or lost to heat-absorbing particles. Turbidities in the Eastern United States in the summer imply net losses to have been about 6 percent. More recent reports from the network (1972-1975) imply net losses of about 10 percent for average summer conditions in the East. Long-term trends in atmospheric turbidity in the Eastern United States are qualitatively consistent with those for airport visibility (Figure 1-14).

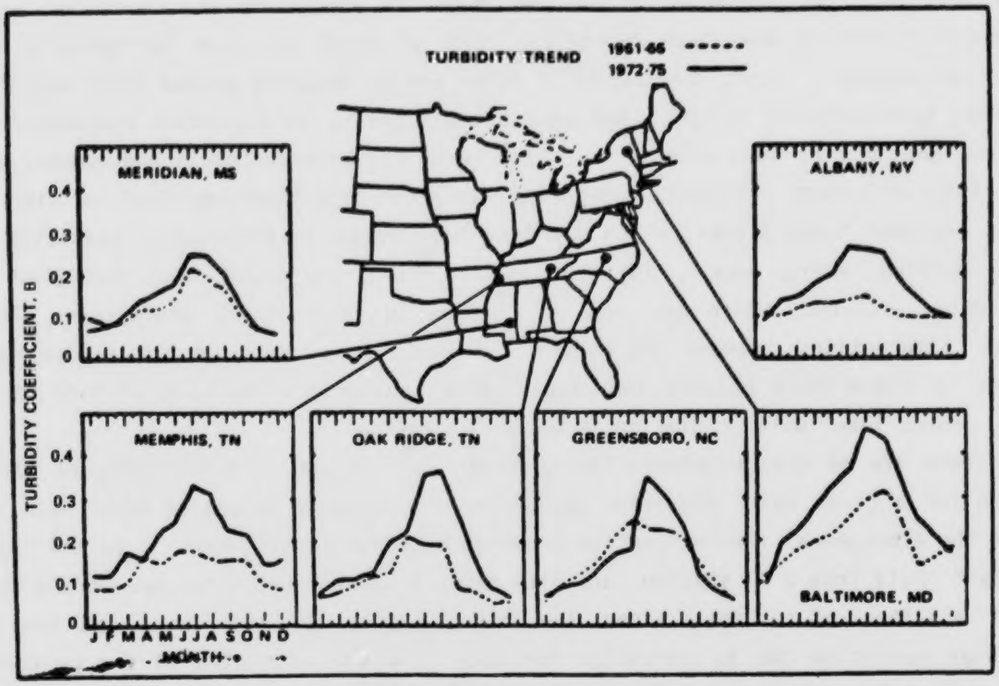


Figure 1-14. Seasonal turbidity patterns for 1961—1966 and 1972—1975 are shown for selected regions in the Eastern United States.

Source: Flowers et al. (1969), and WMO (1974 through 1977).

1.10 EFFECTS ON MATERIALS

Physical damage to materials by sulfur oxides and particulate matter has been investigated in field and laboratory studies. Various approaches have been used to estimate economic damage. Economic determinations have directly related ambient pollutant levels to economic damage estimates, or they have estimated economic damage on the basis of physical damage functions. The latter method, called here the physical damage function approach, has been the method of choice in the past. Other studies, especially in the last decade, have employed the first approach. Both approaches share a common element: an estimation of willingness to pay to reduce the damage.

Approaches employing physical damage functions have been most widely used and, therefore, have received the most extensive treatment. The damage function is a mathematical expression linking exposure to damage, expressed in terms appropriate to the interaction of the pollutant and material. For example, corrosion of metal would be expressed in units of thickness lost, and deterioration of paint in units of reflectance or thickness lost. A major problem in establishing reliable damage functions for a given pollutant involves separating influences of the pollutant from those of meteorological parameters (e.g., temperature, relative humidity, sunlight, windspeed, wind direction) and other air pollutants. For the corrosion of metals, the duration of surface wetness is the most important variable.

Economic valuations may require determinations of a critical damage level. This level represents the point at which the service life or utility of the material ends or is severely impaired. When this point is reached, replacement or repair costs are incurred. For example, if a typical coat of paint is 60 µm thick, the critical damage level at which repainting is necessary might be about 10 µm. Monetary value is determined through economic damage functions, which may be developed from physical damage functions. This approach includes exposure, replacement cost, protection cost, and other data, but it cannot account for damage to irreplaceable items, such as works of art, where the only measurable cost is that of preservation. However, only a few of the functions developed to date are relatively reliable in determining damage, and none has been generally accepted for estimating costs.

The best documented and most significant damages from sulfur oxides and particulate matter involve accelerated corrosion of metal, erosion and soiling of paint, and soiling of buildings and other structures. Erosion of stone and other building materials due to sulfur oxides is also well established, but the importance of sulfur oxides relative to other pollutants is not clear. Although evidence of damage to fibers (e.g., cotton and nylon), paper, leather, and electrical components has been reported, reliable damage functions have not been developed for these materials.

Table 1-2 displays damage functions developed for effects of SO_2 on zinc, steel, and house paint. These equations and the data from which they are derived imply that temperature and particulate matter are relatively unimportant factors in metal corrosion and that the most important factor is surface wetness. Corrosion will not take place when the metal surface is

TABLE 1-2. SELECTED PHYSICAL DAMAGE FUNCTIONS RELATED TO SO_2 EXPOSURE

Material	Reference	Exposure-Response relationships	R^2
Zinc	Haynie and Upham, 1970	$Y = 0.001028\ (RH - 48.8)\ SO_2$	0.92
Galvanized steel	Haynie et al., 1976	$corr = (0.0187\ SO_2 + e^{41.85 - 23{,}240/RT})t_w$	0.91
Galvanized steel	Haynie, 1980	$corr = 2.32\ t_w + 0.0134v^{0.781}SO_2t_w$	Not provided by author.
Oil-base house paint	Spence et al., 1975	$Y = 14.3 + 0.0151\ SO_2 + 0.388\ RH$	0.61
Enameling steel	Haynie and Upham, 1974	$corr = 325\ \sqrt{t}\ e^{[0.00275\ SO_2 - (163.2/RH)]}$	Not provided by authors.
Weathering steel	Haynie et al., 1976	$corr = [5.64\ \sqrt{SO_2} + e^{(55.44 - 31{,}150/RT)}]\ \sqrt{f_w}$	0.91

corr = depth of corrosion or erosion, μm

Y = corrosion/erosion rate, μm/yr

SO_2 = μg/m^3 SO_2

R = gas constant (1.9872 cal/gm mol K)

RH = percent annual average relative humidity

f_w = fractional time of panel wetness

t_w = time of wetness in years

v = wind velocity in m/s

T = geometric mean temperature of panels when wet, K

t = time exposure, years

Note: 1 ppm SO_2 = 2620 μg/m^3.

dry. This dominant factor is usually approximated by a "time-of-wetness" term, that is, the amount of time the relative humidity (RH) exceeds some critical level (60 to 80 percent RH), which varies for different metals. There are, of course, several sources of moisture (e.g., rain, snow, fog, condensation), but RH is the usual proxy for moisture from all sources. Corrosion initiated by surface wetness is accelerated by SO_2. An increase in either the concentration of SO_2 or the relative humidity is accompanied by an increase in the rate of corrosion. The relative importance of the two factors in accelerating corrosion is shown in Figures 1-15 and 1-16, based on analyses of field data. As shown in Figure 1-16, a 100-percent increase in the average concentration of sulfur dioxide has about the same effect as a 10-percent increase in relative humidity above a critical humidity level. In some areas of the country (see Figure 1-17) the humidity is usually at or above the critical level of 60 to 80 percent RH; in other areas, the critical level is rarely reached.

The impact of relative humidity must be taken into account in estimating nationwide damage to metals from SO_2. Average annual RH can vary 10 percent even within one region of the country; for instance, included in the data base for Figures 1-15 and 1-16 are average RH's of 29 and 39 percent for Las Vegas and Phoenix, respectively. The range in RH for 57 sites covering 34 States and the District of Columbia was 29 to 76 percent. Average SO_2 concentrations measured at these sites ranged from 9 to 374 $\mu g/m^3$ (0.003 to 0.14 ppm) during the same period. This wide variation is useful in regression analysis for developing damage functions, but it greatly complicates estimation of aggregate damage. Relative humidity and SO_2 concentrations vary spatially and seasonally. Their respective spatial and temporal variations, however, are not the same across the country. In some areas, the highest SO_2 concentrations coincide with periods of highest relative humidity; in other areas, the reverse is true.

Even if there were a means to predict with perfect precision the amount of corrosion incurred by a metal surface due to a specified level of SO_2, it would still be difficult to arrive at an acceptable aggregate damage estimate. One would have to know the total thickness of the metal layer in question, the critical thickness below which repair or replacement is necessary, and the total area of surface exposed. This information has not been compiled; instead, various surrogates have been used, typically annual production data modified by some service-life factor. These surrogates do not account for such influences as indoor versus outdoor use, use of protective coatings, or subjective judgments as to the point at which the object in use should or could be repaired or replaced. The latter judgment is influenced both by willingness to pay and ability to pay, and is tied to the economic status of the user or owner. Because of these complex factors, recent attempts to relate atmospheric pollutant levels to economic damage have focused on the development of direct relationships between pollutant concentrations and economic benefit or loss of utility.

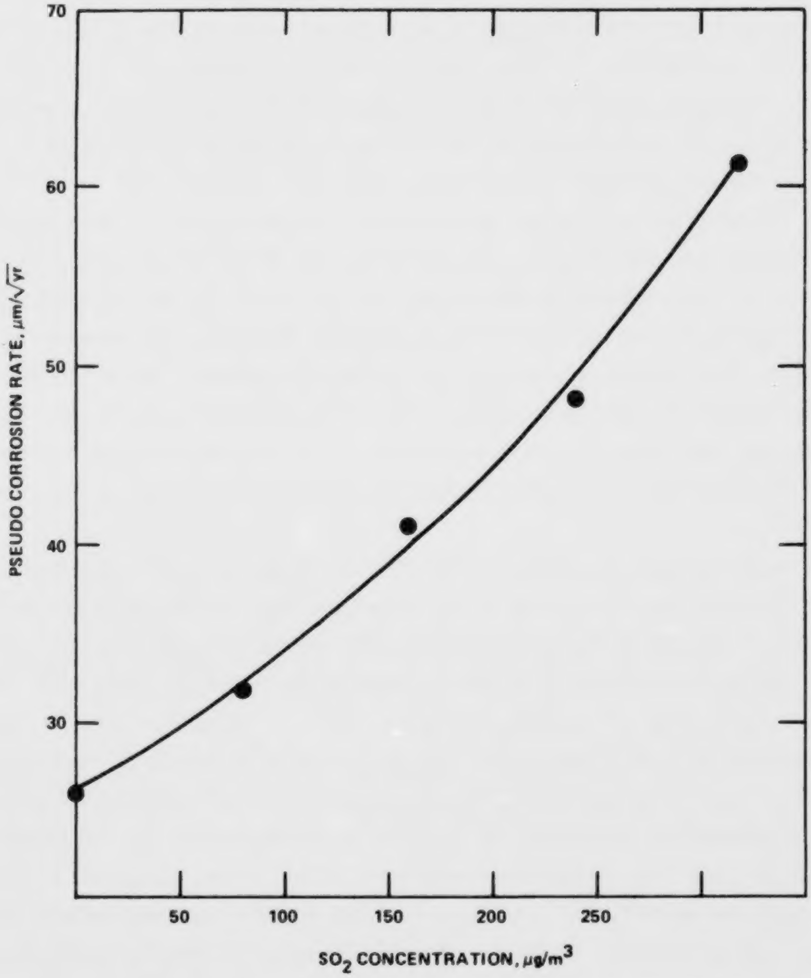


Figure 1-15. Steel corrosion behavior as a function of average SO_2 concentration at 65% relative humidity.

Source: Adapted from Haynie and Upham (1974).

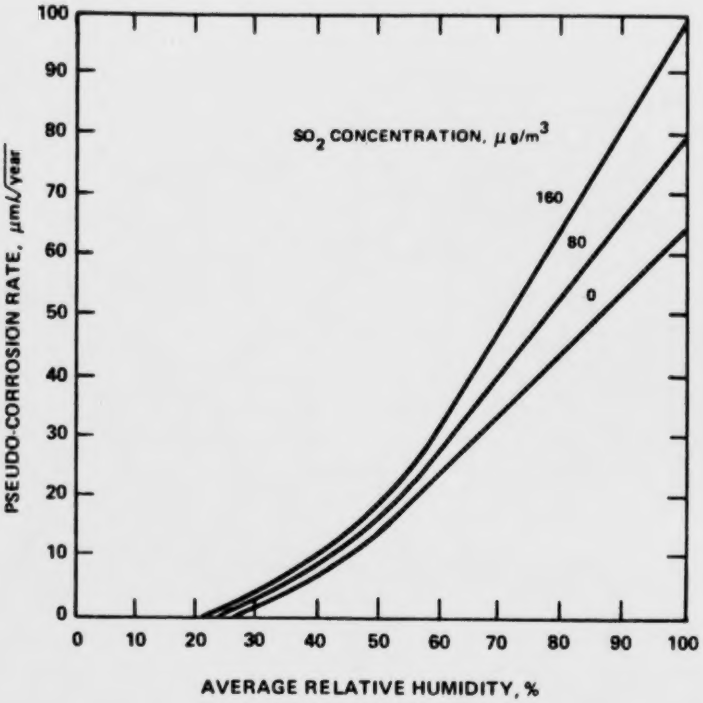


Figure 1-16. Steel corrosion behavior as a function of average relative humidity at three average concentration levels of sulfur dioxide.

Source: Haynie and Upham (1974).

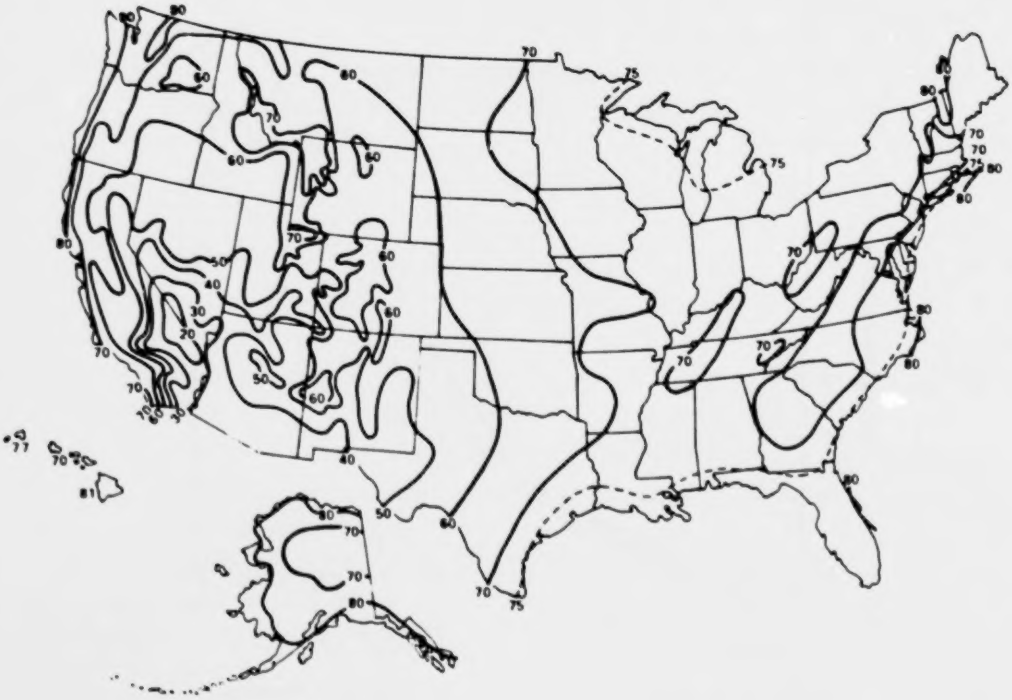

80
60
70
75
50
40
30
20
77
81

Figure 1-17. Isopleths of annual mean relative humidity in the United States.

Source: U.S. Environmental Protection Agency (1979).

Estimates of nationwide erosion of paint and building materials are limited by the same kinds of factors limiting estimates of metal corrosion. Factors such as humidity, nature and extent of exposure, and critical damage points are quantifiable for the nation as a whole only with a great deal of uncertainty. Costs assigned to repair or replacement are often necessarily arbitrary. Furthermore, damage functions for paint and building materials are not as well documented as are those for metals.

The least reliable of the damage functions are those for soiling from PM. Regression analyses show that the relationship between soiling (loss of reflectance) of building materials, including paint, and TSP exposure is tenuous. (See Chapter 3 for information on the lack of a consistent general relationship between reflectance and TSP). Furthermore, reflectance is not the only property of PM important to soiling; also important is particle size, for the deposition velocity of particles depends on their size. Characteristics such as stickiness, oiliness, and tarriness, which would increase adherence of deposited particles to a surface, should also be considered. There is at present no single technique that combines all relevant measurements of reflectance, adherence, and particle size.

The limitations of these and other physical-damage functions hinder accurate estimates of total material damage and soiling. Coupled with these limitations is the lack of material-exposure estimates. These problems currently preclude complete and accurate estimates of the costs of damage based on a physical-damage function approach. Nevertheless, the best estimate of economic loss in 1970 due to SO_2-related materials damage is $900 million (1978 dollars). Reduction in SO_2 levels from 1970 to 1978 are estimated to have resulted in an annual benefit of up to $400 million (1978 dollars) in reduced materials damage. Because of the above-cited difficulties, most soiling-cost estimates developed since 1970 have departed from the physical damage function approach to examine cost and frequency of household cleaning and maintenance tasks. The evidence to date indicates that though cleaning and maintenance expenditures are not a function of TSP levels, the monetary value of increased cleanliness resulting from TSP reduction can be calculated, based on an estimated economic loss of $2000 million (1978 dollars) in 1970 due to exterior household soiling. Reductions in TSP levels from 1970 to 1978 are judged to have resulted in annual benefits of $200 to $700 million from less soiling.

The above estimates for SO_2 and TSP reductions are quite rough, but they can serve to represent the direction and magnitude of changes in benefits associated with improved air quality. Other estimates of costs for materials damage and soiling have related ambient pollutant concentrations directly to economic benefit or loss of utility. The value of such approaches is currently limited by their inability to distinguish the different types of effects of a pollutant, the relative roles of different pollutants, and the influence of socioeconomic variables. Though they show promise for future application, it is not clear at present that these approaches are adequate for decisionmaking guidance.

1.11 RESPIRATORY TRACT DEPOSITION AND FATE OF SULFUR OXIDES AND PARTICULATE MATTER

Information on the deposition and fate of sulfur oxides and particulate matter in various regions of animal and human respiratory systems aids in understanding the findings from animal toxicological, human clinical, and epidemiological studies discussed in subsequent chapters of this document. In both animal laboratory and human clinical studies, exposure levels can usually be measured near the point of inhalation. Moreover, animal studies can often determine the relationship between exposure level and the amount actually reaching the target organ. The monitoring instruments used in these studies, however, vary considerably from those used for ambient air sampling. The resulting differences in exposure characterizations have important implications for quantifying the dose-response relationships that may be derived from the various types of studies described in the health-effects chapters (12, 13, 14) of this document.

The respiratory tract (Figure 1-18) includes the passages of the nose, mouth, nasal pharynx, oral pharynx, epiglottis, larynx, trachea, bronchi, bronchioles, and small ducts and alveoli of the pulmonary acini. With respect to respiratory tract deposition and clearance of inhaled aerosols, three regions can be considered: (1) extrathoracic (ET), the airways extending from the nares down to the epiglottis and larynx at the entrance to the trachea (the mouth is included in this region during mouth breathing); (2) tracheobronchial (TB), the primary conducting airways of the lung from the trachea to the terminal bronchioles (i.e., that portion of the lung respiratory tract having a ciliated epithelium); and (3) pulmonary (P), the parenchymal airspaces of the lung, including the respiratory bronchioles, alveolar ducts, alveolar sacs, atria, and alveoli (i.e., the gas-exchange region). The extrathoracic region, as defined above, corresponds exactly to the definition of the nasopharynx used by the International Commission on Radiological Protection Task Group on Lung Dynamics (Morrow et al., 1966).

The nose is a complex structure of cartilage and muscle supported by bone and lined with mucosa. The vestibule of the nares is unciliated but contains a low-resistance filter consisting of small hairs. The nasal volume is separated into two cavities by a 2- to 7-mm thick septum. The inner fossae and turbinates are ciliated, with mucus flow in the direction of the pharynx. The turbinates are shelf-like projections of bone covered by ciliated mucous membranes with a high surface-to-volume ratio that facilitates humidification of the incoming air. The larynx consists of two pairs of mucosal folds that narrow the airway.

The trachea, an elastic tube supported by 16 to 20 cartilaginous rings that circle about three-fourths of its circumference, is the first and largest of a series of branching airway ducts. The left and right lungs are entered by the two major bronchi that branch off of the trachea (Figure 1-18). The left lung consists of two clearly separated lobes, the upper and lower lobes; the right lung consists of three lobes, the upper, middle, and lower lobes. The conductive airways in each lobe of the lung consist of up to 18 to 20 dichotomous branches from the bronchi to the terminal bronchioles.

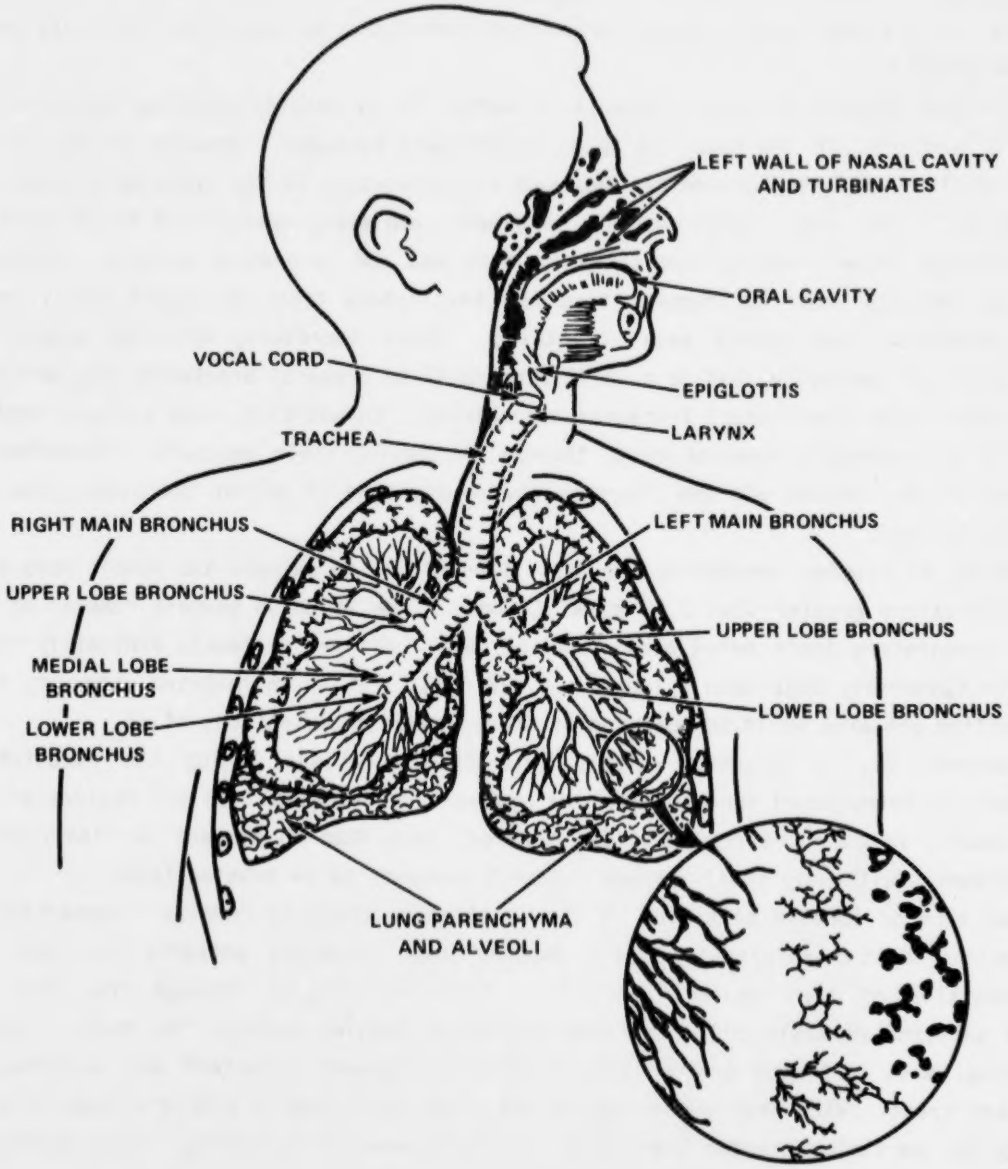

LEFT WALL OF NASAL CAVITY AND TURBINATES
ORAL CAVITY
VOCAL CORD
EPIGLOTTIS
TRACHEA
LARYNX
RIGHT MAIN BRONCHUS
LEFT MAIN BRONCHUS
UPPER LOBE BRONCHUS
UPPER LOBE BRONCHUS
MEDIAL LOBE BRONCHUS
LOWER LOBE BRONCHUS
LOWER LOBE BRONCHUS
LUNG PARENCHYMA AND ALVEOLI

Figure 1-18. Features of the respiratory tract of man used in the description of the deposition of inhaled particles and gases with insert showing parts of a silicon rubber cast of a human lung showing some separated bronchioles to 3 mm diameter, some bronchioles from 3 mm diameter to terminal bronchioles, and some separated respiratory acinus bundles.

Source: Adapted from Hatch and Gross (1964) and Raabe (1979).

The pulmonary gas-exchange region of the lung begins with the partially alveolated respiratory bronchioles. Pulmonary branching proceeds through a few levels of respiratory bronchioles to completely alveolated ducts and alveolar sacs. Alveoli are thin-walled polyhedral air pouches that cluster about the acini through connections with respiratory bronchioles, alveolar ducts, or alveolar sacs. Oxygen uptake and carbon dioxide excretion occur via cells located in the alveoli.

Because sulfur dioxide is highly soluble in water, it is readily absorbed upon contact with the moist surfaces of the nose and upper respiratory passages. Removal of SO_2 by the upper airways during inhalation determines how much SO_2 penetrates to the tracheobronchial and pulmonary regions of the lung. Sulfur dioxide is almost completely removed (95 to 99 percent) by nasal absorption under resting conditions in both man and laboratory animals. However, animal studies indicate that SO_2 removal from the respiratory tract is significantly lower during oral breathing than during nasal breathing. Since increasing activity levels and respiratory workloads generally lead to a shift from nasal to oronasal breathing, SO_2 penetration to the lower respiratory tract increases accordingly. In addition, some persons tend to breathe orally or oronasally even at rest, thereby increasing their exposure. Furthermore, fine particles in the inhaled air may increase the penetration of sulfur compounds into the lower respiratory tract.

The majority of studies concerning the deposition of SO_2 in animals and people have been done at concentrations greater than 2.62 mg/m^3 (1 ppm). The 95 to 99 percent removal of SO_2 by the upper respiratory tract noted above has not been confirmed at levels ordinarily found in ambient air (generally less than 0.1 mg/m^3 [0.038 ppm]). It is anticipated, however, that similar deposition patterns would be observed at these lower concentrations of SO_2.

Once inhaled, SO_2 is absorbed quickly into the secretions lining the respiratory passages. Most is transferred rapidly into the systemic circulation from all regions of the respiratory tract. However, of the total SO_2 inhaled, less than 15 percent is likely to be exhaled immediately, with only small amounts (about 3 percent) to be exhaled later.

The deposition of inhaled particles in the respiratory tract is complex. Deposition in different regions of the respiratory tract depends upon breathing patterns and upon the physical properties of the inhaled particles. Particles inhaled through the nose are deposited in patterns markedly different from particles inhaled through the mouth. During nasal breathing, most particles greater than 4 μm in aerodynamic diameter* are deposited in the respiratory tract. With oral breathing, on the other hand, nearly complete deposition is observed only for particles greater than 10 μm. In both modes of breathing, larger particles are deposited mainly in the upper airways above the trachea. However, 20 to 30 percent of particles between 5 and 10 μm inhaled during oral breathing are deposited farther down the respiratory tract in the trachea and bronchial airways. At levels of light physical activity, only about 10 percent of particles as large as 15 μm are thought to be deposited in the tracheobronchial region.

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*Aerodynamic diameter is defined as the diameter of a unit density sphere having the same settling speed under gravity as the particle in question, whatever its shape and density. Unless stated otherwise in this chapter, all particle sizes are given as aerodynamic diameters.

The deeper penetration of particles into the respiratory tract when a person breathes through the mouth is reflected in the deposition data of Figure 1-19. Generally, between 10 and 20 percent (see lighter shaded area of figure) of inhaled particles less than about 1 μm are deposited in the pulmonary region. About 20-percent pulmonary deposition is typical for particles 1 to 4 μm when inhaled through the nose; when particles are inhaled through the mouth, substantially greater (20 to 70 percent) pulmonary deposition results, especially for particles around 3.5 μm. For nasal breathing, the peak of the pulmonary deposition curve shifts downward from 3.5 to 2.5 μm. Depending upon breathing frequency and the volume of air inhaled or exhaled during a breathing cycle (tidal volume), pulmonary deposition of 5-μm particles can vary from as little as 5 percent to as much as 50 percent during oral breathing. However, only about 5 to 13 percent of particles as large as 8 to 9 μm are deposited in the pulmonary region.

Studies of the regional deposition of particles less than 3 μm have been conducted using dogs, rats, and hamsters. In these animals the relative distribution of the particles along the respiratory tract during nasal breathing follows a regional deposition pattern that is similar to that found in humans during nasal breathing. Hence, it is possible, with appropriate corrections, to extrapolate from these animals to humans for particles in this size range.

Children are usually considered to be more susceptible than adults to the effects of environmental pollutants, but deposition data for children are not currently available, nor are they likely to be obtained soon because of ethical and other constraints on using children as experimental subjects. The few data that are available on other populations, such as persons with asthma or chronic bronchitis, indicate that tracheobronchial deposition appears to increase while pulmonary deposition decreases in most abnormal respiratory conditions.

By taking into account the biological effects of the particles and the population at risk, air-sampling procedures can be formulated to focus on the region or regions of the respiratory tract pertinent to accurate health assessment. The particle-collection characteristics of certain standard samplers intended to reflect selective deposition patterns in different regions of the respiratory tract are depicted in Figure 1-19.

Particles deposited in different regions of the respiratory tract are cleared by different pathways and at different rates. Particles deposited in the anterior regions of the nasal passages are cleared forward by nose-blowing and sneezing. Beyond the middle turbinate

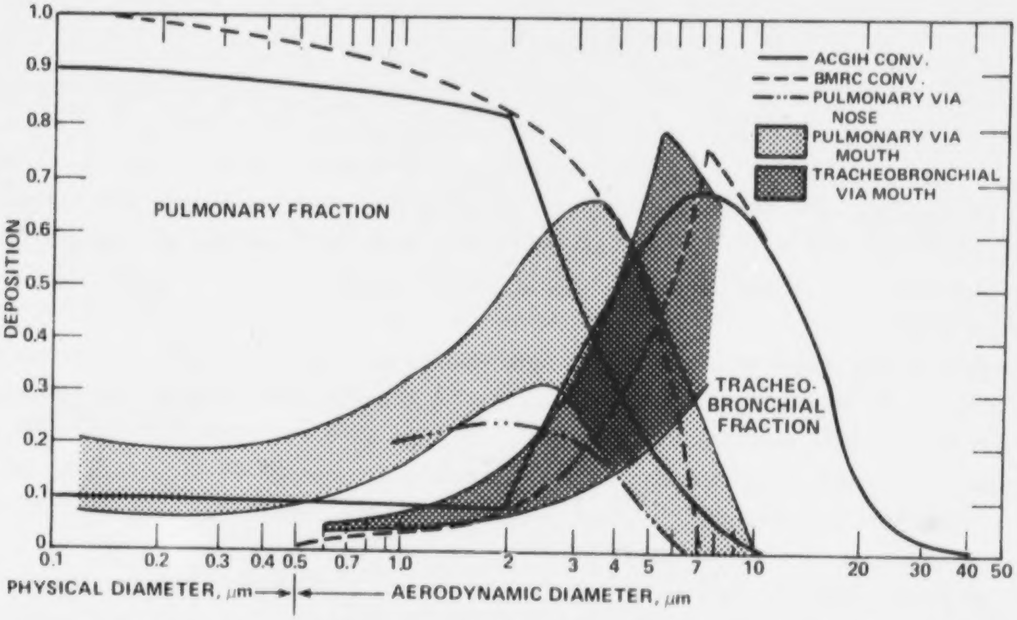

Figure 1-19. Division of the thoracic fraction of deposited particles into pulmonary and tracheobronchial fractions for two sampling conventions (ACGIH and BMRC) as a function of aerodynamic diameter, except below 0.5μm, where physical diameter is used (International Standards Organization, 1981). Also shown are bands for experimental pulmonary deposition data from Figure 11-9 and for tracheobronchial (TB) deposition as a percent of particles entering the mouth. The band for TB deposition was derived using the overall regression line of Chan and Lippmann (1980) for extrathoracic deposition with oral breathing and their equation for TB deposition, which was evaluated at bronchial deposition size values one standard deviation from the mean, given an average inspiratory flow rate of 30 liters per minute. The TB band is shown up to about the largest particle size used by Chan and Lippmann (1980).

Source: ACGIH (Threshold Limits Committee, 1968); BMRC (Orenstein, 1960); Pulmonary via nose (Lippmann, 1977); Pulmonary via mouth (see Figure 11-9); Tracheobronchial via mouth (Chan and Lippmann, 1980).

region of the nose, clearance to the pharyngeal regions occurs by mucociliary action, after which the particles are generally swallowed. Likewise, most clearance of material deposited in the oral cavity is by swallowing. All of these processes are relatively rapid and remove most of the deposited material from the respiratory tract within minutes to hours.

Insoluble particles deposited in the tracheobronchial region are cleared upward in the respiratory tract by mucociliary action and are then swallowed. This clearance is usually complete within one or two days after deposition. In contrast, particles deposited in the pulmonary region may be retained for several hundred days before they are cleared to the conducting airways or to the pulmonary lymphatic system.

As particles are cleared by mechanical processes from all regions of the respiratory tract, chemical dissolution may remove soluble compounds, which can then be absorbed directly into the systemic circulation. Since dissolution and absorption of substances from particles deposited in the respiratory tract compete with mechanical clearance processes, the amount absorbed depends upon the rate of dissolution as compared to the rate of mechanical clearance. The proportion of deposited material that is absorbed into the body varies markedly for different regions of the respiratory tract because of the large variations in clearance rates.

1.12 TOXICOLOGICAL STUDIES

Toxicological studies of the metabolism and effects of sulfur oxides and various forms of particulate matter in experimental animal subjects are discussed in Chapter 12. Although inhaled sulfur compounds are rapidly absorbed into the systemic circulation, their main effect is observed in the respiratory tract. Prior to or during inhalation, SO_2 may react with water to form sulfurous acid (H_2SO_3) or be oxidized to form sulfur trioxide (SO_3). The latter reacts rapidly with water to form sulfuric acid (H_2SO_4), which subsequently forms ammonium sulfate in the presence of ammonia. Sulfurous acid readily dissociates to sulfite and bisulfite ions, which are in rapid chemical equilibrium. Bisulfite ions react with biological molecules by sulfonation, by auto-oxidation, and by addition to cytosine. Most of the inhaled SO_2 is presumed to be detoxified in the liver and other organs by the sulfite-oxidase pathway, which forms sulfate that can then be excreted in the urine.

The metabolism of toxic substances that may be inhaled with atmospheric particles is specific to the individual compounds. A discussion of the metabolism of all potentially inhalable compounds in urban air is beyond the scope of this document. Detailed studies on the deposition and clearance in laboratory animals of coal combustion products, automobile exhausts, and silicates have been reported elsewhere in the scientific literature.

A number of studies have been conducted on the effects of exposing various species of laboratory animals to different concentrations of SO_2. Some of these studies examined the effects of short-term SO_2 exposures on pulmonary function in animals (see Table 1-3). From these investigations it appears that constriction of the bronchial air passages (bronchoconstriction) is the most likely effect of 1-hour exposure to SO_2; but at levels below 2.62 mg/m^3 (1 ppm) such effects are not consistently observed.

TABLE 1-3. EFFECTS OF ACUTE EXPOSURES TO SULFUR DIOXIDE ON PULMONARY FUNCTION

Source table	Concentration	Duration	Species	Results	Reference
12-3	0.42 or 0.84 mg/m^3 (0.16 or 0.32 ppm)	1 hr	Guinea pig	Increase in airway resistance.	Amdur and Underhill, 1970; Amdur et al., 1978a
12-3	0.52, 1.04, 2.1 mg/m^3 (0.2, 0.4, 0.8 ppm)	1 hr	Guinea pig	No significant increase in airway resistance.	Amdur et al., 1978c
12-3	2.62, 5.24, 13.1, 26.2 mg/m^3 (1, 2, 5, 10 ppm)	1 hr	Dog	Increased bronchial reactivity to aerosols of acetylcholine, a potent bronchoconstrictive agent.	Islam et al., 1972
12-3	0, 44.5, 83.8, 162, 233, 322, 519, 781 mg/m^3 (0, 17, 32, 62, 89, 123 198, 298 ppm)	10 min	Mouse	Respiratory rate decreased proportionally to the log of the concentration. Time for maximum response was inversely related to the log of the concentration; recovery complete within 30 min following all exposures.	Alarie et al., 1973d

It has been hypothesized that SO_2 induces bronchoconstriction by stimulating bronchial epithelial receptors that initiate reflexive contraction of smooth muscles in the bronchial air passages. This reflex is pharmacologically mediated by portions of the autonomic nervous system, particularly the vagus nerve, and apparently involves the release of acetylcholine or histamine. Sulfur dioxide is thought to produce bronchoconstriction in humans also through the same autonomic reflex arc (see Section 1.13).

Since similar, if not identical, bronchoconstrictive effects are produced by histamine aerosols and SO_2, there is some plausibility in the view that histamine may also be involved in the bronchoconstriction initiated by SO_2. Other similarities tend to support this hypothesis. For example, the effects of SO_2 and of histamine aerosols are both seen over broad ranges of concentrations. To wit, as much as a 200-fold difference in dose has been reported for histamine-induced bronchoconstriction. In addition, as in the case of SO_2, histamine sensitivity may decrease with age, depending upon the species. Further studies are needed to substantiate this hypothesis about the role of histamine and to determine if the variation in response to SO_2 represents sensitive populations.

Another alteration in breathing caused by SO_2 is a transient decrease in respiratory rate. This effect may involve a chemoreceptor in the nasal passages (similar to the one thought to be responsible for bronchoconstriction). It also may involve the release of acetylcholine and is thought to be mediated by the trigeminal nerve. The decrease in respiratory rate induced by SO_2 requires a higher concentration than the bronchoconstrictive effect (see Table 1-3) and differs in other respects, among which are a concentration-independent transience and a concentration-dependent period of desensitization.

The primary host-defense mechanism of the respiratory tract is the clearance of foreign objects from the lung, whether by mechanical means (mucociliary transport) or biological means (phagocytosis or immunological processes). The effects of SO_2 on these mechanisms are variable and species-dependent. For example, rats exposed to SO_2 at 0.26 mg/m^3 (0.1 ppm), 7 hours/day, 5 days/week for either 2 or 3 weeks exhibited accelerated clearance of radioisotope-labeled particles 10 days and 23 days after SO_2 exposure terminated. In the same study, 2.6-mg/m^3 (1-ppm) SO_2 exposures also accelerated clearance of particles 10 days later but depressed clearance rates 25 days later. In other studies, mucus flow in the trachea of dogs decreased following intermittent exposure for 1 year to SO_2 at a concentration of 2.62 mg/m^3 (1 ppm), whereas a single 30-minute exposure of 65 mg/m^3 (25 ppm) did not affect clearance in donkeys. Also, from limited work with infectivity models, it appears that susceptibility to bacterial infection is not affected by high concentrations of SO_2 (13 mg/m^3 [5 ppm] for up to 3 months). Antiviral defenses were impaired by SO_2 in mice, but only at exposures to a minimum of 18.3 to 26.2 mg/m^3 (7 to 10 ppm) for 7 days. Chronic exposure to SO_2 (5.2 mg/m^3 [2 ppm] for 192 days), however, can cause alterations of the pulmonary and systemic immune systems. In summary, acute exposure to SO_2 can alter some aspects of host defenses, but concentrations in excess of those currently found in the ambient air appear to be required. Unfortunately, few studies have examined effects of chronic exposures at lower concentrations.

In regard to possible respiratory tract pathology, no remarkable alterations in lung morphology have been observed following chronic exposure to SO_2 in monkeys (0.36 to 13.4 mg/m^3 [0.14 to 5.1 ppm] for 540 days) or dogs (13.4 mg/m^3 [5.1 ppm] for 620 days). However, only conventional light microscopy was used, a method far less sensitive than scanning- or transmission-electron microscopy for observing alterations in surface membranes and cilia. Shorter exposures to much higher concentrations (26 to 1050 mg/m^3 [10 to 400 ppm]) generally did cause morphological changes in mice, rats, and pigs.

The issue of whether SO_2 is a mutagen in humans is currently unresolved. Although mutagenesis in response to SO_2 has been demonstrated in two microorganisms *in vitro* at acidic pH levels, no evidence supports its occurrence in at least two higher systems, viz. *Drosophila* and mouse oocytes.

With regard to the tumorigenic properties of SO_2, *in vivo* investigations of its potential oncogenicity are quite rare. Tumorigenesis has been examined in mice and rats after exposure to SO_2 or to a combination of SO_2 and benzo(a)pyrene, respectively. In one study, mice were exposed to SO_2 intermittently over an entire lifetime. Increased incidence of primary lung carcinoma was reported for females, but not for males. Because an adequate statistical analysis was not presented in the report of the study, a subsequent statistical analysis was performed, revealing that the increase in primary lung carcinoma was significant ($p = 0.011$) in SO_2-exposed females but not males. However, the exact duration of exposure and concentration cannot be determined accurately from the published report.

The effects of lifetime simultaneous exposure to SO_2 (10.5 mg/m^3 [4 ppm]) and benzo(a)pyrene (10 mg/m^3) were studied in rats. The biological significance of this study is difficult to interpret, particularly since statistical analyses were not given. However, subsequent statistical analysis of the data reported for a combined exposure revealed that the increased incidence of lung tumors was statistically significant ($p = 0.005$), whereas the effects of exposure to SO_2 and to benzo(a)pyrene alone were not significant.

Numerous animal studies have investigated mortality induced by sulfur dioxide. However, SO_2 causes increased mortality only at high concentrations ($\geq$131 mg/m^3 [50 ppm]) that are not relevant to ambient air exposure levels.

Characterizing exposures to particles in the atmosphere may be even more difficult than characterizing exposures to SO_2. This difficulty is due, in large part, to the fact that the toxicity of particulate matter depends greatly upon its chemical composition. In general, urban air is quite heterogeneous in composition and may vary widely from one community to the next, or even within a single community. It may contain both inert and chemically toxic constituents, with the potential impact of the latter being complicated by such considerations as dissolution, solubility, and biological availability. Although adequate physical and chemical information can be obtained from studies with laboratory animals exposed to homogeneous aerosols, the above-mentioned types of data are not available for the heterogeneous mixture of particles found in the environment. Therefore, the comparisons that can be made between toxicological studies of animals exposed to well-defined, laboratory-produced aerosols and epidemiological studies of people exposed to environmental aerosols are extremely limited.

Sulfur compounds that have been used in particulate form in inhalation studies include: sulfuric acid, ammonium sulfate and bisulfate, and sulfate salts of zinc, iron, copper, manganese, and other metals. Alterations of pulmonary function, particularly increases in pulmonary flow resistance, provide a measure of response to acute exposure to aerosols of particulate sulfur compounds. Reports of the irritant potency of various sulfate species are variable, perhaps due to differences in animal species and strains and to different particle sizes, pH, composition, and solubility. Generally, however, sulfuric acid appears to be more irritating than any of the sulfate salts, as reflected in Table 1-4, which ranks various sulfate species in terms of their irritant potency (measured as increased airway resistance), based on short-term (1-hour) exposures. As Table 1-5 indicates, for short-term exposures the lowest concentration of sulfuric acid found to increase airway resistance was 100 μg/m^3. This finding was obtained with guinea pigs, which appear to be more sensitive than other laboratory animal species to aerosols of sulfuric acid.

TABLE 1-4. RELATIVE IRRITANT POTENCY OF SULFATE SPECIES IN GUINEA PIGS EXPOSED FOR ONE HOUR[a]

Sulfuric acid	100
Zinc ammonium sulfate	33
Ferric sulfate	26
Zinc sulfate	19
Ammonium sulfate	10
Ammonium bisulfate	3
Cupric sulfate	2
Ferrous sulfate	0.7
Sodium sulfate (at 0.1 μm)	0.7
Manganous sulfate	-0.9

[a] Data are for 0.3-μm mass median diameter particles. Increases in airway resistance were related to sulfuric acid (0.41 percent increase in resistance per μg of sulfate as sulfuric acid), which was assigned a value of 100.

Source: Amdur et al. (1978a).

The irritant potency of sulfuric acid aerosol depends in part on particle size. As reflected to some extent by the findings reported in Table 1-6, sulfuric acid particles approximately 2 μm or smaller (mass median diameter) generally have more effect on respiratory function. The irritant potency of sulfuric acid may also be affected by its partial neutralization by ammonia present in the breath or in the air of exposure chambers. The resulting

TABLE 1-5. RESPONSES TO ACUTE SULFURIC ACID EXPOSURE

Source table	Concentration	Duration	Species	Results	Reference
12-6	100 $\mu g/m^3$	1 hr	Guinea pig	Pulmonary resistance increased 47%; pulmonary compliance decreased 27%.	Amdur, 1977; Amdur et al., 1978b
12-9	500 $\mu g/m^3$	1 hr	Dog	Slight increases in tracheal mucociliary transport velocities immediately and 1 day after exposure. One wk later clearance was significantly decreased.	Wolff et al., 1979
12-6	510 $\mu g/m^3$	1 hr	Guinea pig	Pulmonary resistance increased 60%; pulmonary compliance decreased 33%.	Amdur et al., 1978b
12-6	1000 $\mu g/m^3$	1 hr	Guinea pig	Pulmonary resistance increased 78%; pulmonary compliance decreased 40%.	Amdur et al., 1978b
12-9	190-1400 $\mu g/m^3$	1 hr	Donkey	Bronchial mucociliary clearance was slowed.	Schlesinger et al., 1978
12-9	1000 $\mu g/m^3$	1 hr	Dog	Depression in tracheal mucociliary transport rate persisted at 1 wk after exposure.	Wolff et al., 1979
12-9	1400 $\mu g/m^3$	1 hr	Donkey	No effect on tracheal transport.	Schlesinger et al., 1978

TABLE 1-6. RESPONSES TO CHRONIC SULFURIC ACID EXPOSURE

Source table	Concentration and particle size*	Duration	Species	Results	Reference
12-9	100 μg/m^3 (0.3-0.6 μm)	1 hr/day, 5 day/wk, 6 mo	Donkey	Within the first few weeks all four animals developed erratic bronchial mucociliary clearance rates, either slower than or faster than those before exposure; animals not exposed to H_2SO_4 before this study had slowed clearance during the second 3 mo of exposure and for 3-4 mo after the end of of the exposure.	Schlesinger et al., 1978
12-5 12-8	80 μg/m^3 (0.84 μm); 100 μg/m^3 (2.78 μm)	52 wk cont.	Guinea pig	No significant blood effect; no lung alterations; no effect on pulmonary function.	Alarie et al., 1973a, 1975
12-5 12-8	380 μg/m^3 (1.15 μm); 480 μg/m^3 (0.54 μm)	78 wk cont.	Monkey	No significant blood effect; 380 μg/m^3 increased respiratory rate; 480μg/m^3 had no effect on respiratory rate but altered distribution of ventilation early in exposure period but not later.	Alarie et al., 1973a
12-13	900 μg/m^3 (2.78 μm)	52 wk cont.	Guinea pig	No significant effects on hematology, pulmonary function, or morphology.	Alarie et al., 1975

*All particle sizes in mass median diameter (geometric median size of a distribution of particles, based on weight).

ammonium sulfate and ammonium bisulfate lessen the effects of sulfuric acid, but the extent to which they have affected the results of available animal studies cannot be quantified.

As summarized in Tables 1-5, 1-6, and 1-7, chronic exposure effects of sulfuric acid and sulfate salts are less certain than acute exposure effects. Exposure to sulfuric acid at a concentration of 380 $\mu g/m^3$ for 78 weeks was reported to produce small increases in respiration rate in monkeys, but measurable respiration-rate changes did not occur at a concentration of 480 $\mu g/m^3$. Duration of exposure, concentration, particle size, and chemical composition all appear to be important in determining changes in pulmonary function.

Exposure to sulfuric acid mist causes an alteration in mucociliary clearance of viable as well as nonviable bacterial particles from the lung. The effects are variable. For example, tracheal mucociliary transport in dogs increased after exposure to sulfuric acid aerosol at 500 $\mu g/m^3$ for 1 hour but decreased in rats and hamsters after 1- to 3-hour exposures to 1000 $\mu g/m^3$. Prolonged daily 1-hour exposures of donkeys to 100 $\mu g/m^3$ of 0.5-μm sulfuric acid aerosols caused a persistent slowing of mucociliary transport.

Resistance to bacterial infection is not affected by exposure to sulfuric acid mist. However, various metal sulfates adversely affect this defense mechanism. The potency of these metal sulfates, based on a 3-hour exposure causing an increased susceptibility to bacterial infection, may be ranked as: cadmium sulfite > cupric sulfate > zinc sulfate > aluminum sulfate > zinc ammonium sulfate. At concentrations greater than 2.5 mg/m^3, sulfuric acid and the following sulfates were ineffective in this bacterial infection paradigm: ammonium sulfate, ammonium bisulfate, sodium sulfate, ferric sulfate, and ferric ammonium sulfate. It should be noted, however, that various nonsulfate metallic aerosols, especially compounds containing nickel or cadmium, have substantial inhibitory effects on host-defense mechanisms in general.

Changes in pulmonary morphology due to particulate matter have been studied mostly after chronic exposure to sulfuric acid. Morphological changes were evident in the lungs of monkeys after a long-term (78-week) exposure to relatively high levels of sulfuric acid ($>2.43\ mg/m^3$). The major findings included thickening of the bronchial wall and bronchiolar epithelium, which may contribute to changes in lung function. In other studies, involving chronic exposure of guinea pigs (for 1 year) and dogs (for about 2 years) to sulfuric acid, neither morphological nor physiological changes were noted at concentrations of less than 1 mg/m^3.

The lethal effects of sulfate aerosols depend in part on an animal's age. For example, 18 mg/m^3 sulfuric acid was lethal to 1- to 2-month-old guinea pigs, as opposed to 50 mg/m^3 for 18-month-old animals. Particle size is also important: for guinea pigs the LC_{50}* was 30 mg/m^3 with particles of 0.8-μm diameter, as opposed to 109 mg/m^3 with particles of 0.4-μm diameter. Bronchial spasm appeared to be the major cause of death.

Suspended particles not related to sulfur oxides are also of concern. However, because of the wide variety of such substances, it is difficult to summarize pertinent toxicological results. Information on the inhalational toxicology of several individual substances found in

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*LC_{50} refers to the lethal concentration of a substance for 50 percent of tested subjects.

TABLE 1-7. RESPONSES TO VARIOUS PARTICULATE MATTER MIXTURES

Source table	Concentration	Duration	Species	Results	Reference
12-6	100 μg/m³ open hearth dust	1 hr	Guinea pig	Pulmonary resistance increased 9%; no change in pulmonary compliance.	Amdur and Underhill, 1968, 1970
12-6	500 μg/m³ $(NH_4)_2SO_4$	1 hr	Guinea pig	Pulmonary resistance increased 23%; pulmonary compliance decreased 27%.	Amdur et al., 1978a
12-6	750 μg/m³ Na_3VO_4	1 hr	Guinea pig	Pulmonary resistance increased 7%.	Amdur and Underhill, 1968
12-6	1000 μg/m³ $FeSO_4$	1 hr	Guinea pig	Pulmonary resistance increased 2%.	Amdur and Underhill, 1968
	1000 μg/m³ $MnCl_2$	1 hr	Guinea pig	Pulmonary resistance increased 4%.	Amdur and Underhill, 1968
12-10	75-1940 μg/m³ Cd as $CdCl_2$ 100-670 μg/m³ Ni as $NiCl_2$ 500-5000 μg/m³ Mn as Mn_3O_4	2 hr	Mouse	Increased mortality from subsequent standard airborne streptococcal infection at: 100 μg/m³ for $CdCl_2$, 500 μg/m³ for $NiCl_2$, and 1550 μg/m³ for Mn_3O_4.	Gardner et al., 1977b Adkins et al., 1979, 1980
12-13	1500 μg/m³ carbon; H_2SO_4 + 1500 μg/m³ carbon	3 hr/day 5 day/wk, 20 wk	Mouse	Immune system altered. Morphological changes observed; more severe with exposure to carbon alone.	Fenters et al., 1979
12-13	1100 μg/m³ H_2SO_4, or 1500 μg/m³ carbon, or combination	3 hr	Hamster	No change in ciliary beat frequency after carbon exposure; frequency depressed after H_2SO_4 exposure. Combination produced similar effects, but recovery occurred by 48 hr postexposure. Up to 48 hr after exposure, H_2SO_4 + carbon resulted in more tissue destruction than either pollutant alone.	Schiff et al., 1979

ambient air particulate matter is summarized in Table 1-7. Relevant information can also be derived from noninhalational toxicological studies. For example, numerous trace metals have been found as components of airborne particulate matter. In addition to being generally toxic, certain compounds of some of these metals, including arsenic, beryllium, cobalt, and nickel, have been identified as carcinogenic under specific, nonrespiratory laboratory-exposure conditions.

Silicon is ubiquitous in the earth's crust and in airborne coarse-mode particles. Silicon dioxide (SiO_2), which is responsible for the disease silicosis, is found in three crystalline forms, which occur in the following general order of toxicity: tridymite > cristobalite > quartz. These uncombined forms of SiO_2 are generally called "free silica." Silicon dioxide is also found combined with cations in silicates. There have been few animal toxicological studies of silicates. Several hypotheses on the etiology of silicosis have been developed, but none has been proven. Although many animal toxicological studies of SiO_2 exist, comparisons are difficult because of differences in the species and strains of animals used, the occurrence of accidental infections, and the variations in size and crystalline form of the SiO_2 particles. Silicosis similar to that observed in man has been produced in animals exposed to high concentrations of quartz and other SiO_2 dusts via intratracheal instillation (30-50 mg) or chronic inhalation. Chronic (2.5-year) exposures of dogs to aerosols of diatomaceous earth containing 61-percent cristobalite produced fibrotic nodules in hilar lymph nodes, but not in the lungs.

It is difficult to assess with accuracy the toxicity of complex sulfur-containing aerosols in urban atmospheres based simply upon their sulfuric acid or sulfate content. The chemical composition of aerosol sulfate compounds, particularly the metal or cation component, is important in determining their relative toxicity. Since atmospheric aerosols may contain varying proportions of sulfuric acid and ammonium or metal sulfates, it is not possible to extrapolate from animal toxicological data obtained with single compounds to ambient environmental conditions.

Liquid or solid particles, which may act as carriers for SO_2, seem to enhance the toxic effects of the gas in some cases. Table 1-8 shows examples where the aerosols contain solutions of salts such as sodium chloride (NaCl), manganese chloride ($MnCl_2$), or ferrous sulfate ($FeSO_4$). Although the evidence is not clear, synergistic as well as additive toxic effects have been observed by some investigators using sulfuric acid and ozone (see Table 1-9). In addition, conversion of SO_2 to H_2SO_4 may increase the toxic potency of SO_2. On the other hand, it may be seen from Table 1-10 that the addition of fly ash to mixtures of SO_2 and H_2SO_4 had no significant effect.

TABLE 1-8. RESPONSES TO ACUTE EXPOSURE COMBINATIONS OF SO_2 AND SOME TYPES OF PARTICULATE MATTER

Source table	Concentration	Duration	Species	Results	References
12-11	2.62 μg/m^3 (1.0 ppm) SO_2 + 1 mg/m^3 NaCl aerosol (<40% RH and >80% RH)	1 hr	Guinea pig	No increase in pulmonary flow resistance at low RH; at high RH, potentiation was marked and evident during both early and late parts of exposure.	McJilton et al., 1973
12-11	2.62 μg/m^3 (1.0 ppm) SO_2 + Aerosols of various salts	1 hr	Guinea pig	Presence of soluble salts increased pulmonary flow resistance about threefold. The potentiation was evident early in the exposure.	Amdur and Underhill, 1968
12-11	5.24 μg/m^3 (2.0 ppm) SO_2 + 4, 10 mg/m^3 NaCl aerosol	1 hr	Guinea pig	SO_2 alone produced an increase of 20% in pulmonary flow resistance; with 10 mg/m^3 NaCl the increase was 55% and the potentiation occurred later in exposure; with 4 mg/m^3 potentiation was reduced.	Amdur, 1961

TABLE 1-9. RESPONSES TO ACUTE EXPOSURE COMBINATIONS OF SULFURIC ACID AND OZONE

Source table	Concentration	Duration	Species	Results	Reference
12-14	880 μg/m^3 H_2SO_4 + 0.1 ppm O_3	3 hr O_3 2 hr H_2SO_4	Hamster	H_2SO_4 depressed ciliary beat frequency; by 72 hr after exposure, recovery had occurred. O_3 exposure had no effect. Sequential exposure to O_3 then H_2SO_4 decreased ciliary beat frequency significantly but to a lesser extent than that caused by H_2SO_4 alone.	Grose et al., 1980
12-14	900 μg/m^3 H_2SO_4 + 0.1 ppm O_3	3 hr O_3 2 hr H_2SO_4	Mouse	Significant increase in mortality in response to airborne infections only when O_3 was given immediately before exposure to H_2SO_4, in which case the effect was additive.	Gardner et al., 1977a
12-14	1000 μg/m^3 H_2SO_4 + 0.4-0.5 ppm O_3	3 days cont.	Rat	Synergistic effects. Glycoprotein synthesis was stimulated in tracheal ring explants; lung DNA, RNA and protein content increased.	Last and Cross, 1978

TABLE 1-10. PATHOLOGICAL RESPONSES FOLLOWING CHRONIC EXPOSURE TO SO_2 ALONE AND IN COMBINATION WITH PARTICULATE MATTER

Source table	Concentration	Duration	Species	Results	Reference
12-2	26.2 mg/m³ (10 ppm) SO_2	72 hr cont.	Mouse	Pathological changes in the nasal mucosa appeared after 24-hr exposure and increased in severity after 72-hr exposure. Mice free of upper respiratory pathogens were significantly less affected than the control animals. Morphological alterations were qualitatively identical in both groups.	Giddens and Fairchild, 1972
12-2	0.37, 1.68, 3.35 mg/m³ (0.14, 0.64, 1.28 ppm) SO_2	78 wk cont.	Cynomolgus monkey	No remarkable morphological changes in the lung.	Alarie et al., 1972, 1973c
12-13	0.29, 2.62, 13.1 mg/m³ (0.11, 1.0, 5.0 ppm) SO_2 + 560 µg/m³ fly ash	78 wk cont.	Cynomolgus monkey	No effects on morphology.	Alarie et al., 1973b
12-13	2.62 mg/m³ (1.0 ppm) SO_2 + 1000 µg/m³ H_2SO_4 (MMD=0.5 µm) + 500 µg/m³ fly ash (MMD=5 µm)	18 mo	Cynomolgus monkey	No significant effects on hematology or pulmonary function during exposure. At end of exposure to SO_2 + H_2SO_4 lungs had morphological alterations in the bronchial mucosa. Exposure to SO_2 + H_2SO_4 + fly ash produced similar alterations; thus fly ash did not enhance effect. Exposure to H_2SO_4 + fly ash produced only slight alterations.	Alarie et al., 1975
12-13	0.29, 2.62, 13.1 mg/m³ (0.11, 1.0, 5.0 ppm) SO_2 + 560 µg/m³ fly ash	52 wk	Guinea pig	No effects on morphology.	Alarie et al., 1973b
12-13	13.4 mg/m³ (5.1 ppm) SO_2 + 900 µg/m³ H_2SO_4	21 hr/day 620 days	Dog	After 225 days, dogs receiving H_2SO_4 had a lower diffusing capacity for CO than those that did not receive H_2SO_4. No morphological changes after 620 days. H_2SO_4 decreased net lung volume and total weight.	Lewis et al., 1969, 1973

1.13. CONTROLLED HUMAN EXPOSURE STUDIES

Chapter 13 discusses clinical studies of the effects of sulfur dioxide and particulate matter on humans. Such studies provide a necessary bridge between epidemiological and animal toxicological data for characterizing health effects induced by air pollution. Unlike community epidemiological studies that investigate health responses of large population groups under highly variable ambient exposure conditions, controlled human exposure (clinical) studies typically evaluate much smaller numbers of subjects but under much better defined and carefully controlled exposure conditions. In the latter type of studies, exposures to either single pollutants or combinations of pollutants are usually carried out in environmentally controlled chambers in which relative humidity, temperature, and pollutant concentrations are designed to approximate representative ambient air exposure conditions, especially those thought to be associated with the induction of acute effects.

Generally inherent in the design of controlled human exposure studies carried out in the United States are limitations on the range or types of pollutant exposures and types of subjects studied so as to assure (as approved by human rights and medical ethics committees) that the experimental exposures to the pollutants being tested per se will not lead to serious morbidity, irreversible illness, or death. Consequently, the types of pulmonary responses assessed in controlled exposure studies are typically "transient" and "reversible." However, depending upon the population at risk, the method of exposure, and the level of subject activity, the so-called mild and reversible health effects measured in controlled human exposure studies may be indicators of other, more serious, associated health effects likely to occur if more prolonged or repeated ambient exposures to the same concentrations of pollutants were encountered by study subjects; or the observed effects per se may be sufficient to interfere with normal work or social activities of certain individuals under some ambient circumstances. For example, relatively small increases in airway resistance of no particular health concern for healthy, nonsensitive adults may be of medical importance for asthmatic individuals or other sensitive groups with already compromised pulmonary functions, especially when accompanied by symptoms associated with or indicative of the onset of more severe breathing difficulties for them under ambient conditions.

In general, the population groups at special risk to air pollution include the young, the elderly, and individuals predisposed by some particular disease, such as asthma, bronchitis, cystic fibrosis, emphysema, and cardiovascular disease. In the normal population, there are also nondiseased but hypersensitive individuals. Such nondiseased "hyperreactors" have been found among at least three of the distinct population groups (normal, chronic bronchitic, and asthmatic) that have been evaluated under controlled exposure conditions in regard to their responses to SO_2 and particulate matter (Lawther, 1955; Frank et al., 1964; Nadel et al., 1965; Burton et al., 1969; Lawther et al., 1975; Jaeger et al., 1979; Sheppard et al., 1980, 1981; Stacy et al., 1981).

In evaluating responses of the above population groups, various investigators have assessed the effects of varying the activity levels of the subjects, the mode of exposure (e.g., nasal, oral, oronasal, or open chamber), and the duration of exposure. One purpose of increasing the activity level during exposure is to simulate outdoor exposures during daily activities by increasing minute ventilation (Ve), i.e., the volume of air expired in one minute. A large majority of normal subjects at rest breathe almost exclusively through the nose with a Ve of approximately 5 to 10 liters. However, some healthy individuals may have abnormally obstructed nasal passages or, for other reasons, regularly breathe oronasally even at rest (Niinimaa, 1980, 1981; D'Alfonso, 1980) Also, certain population groups at risk (such as those with asthma) include some individuals who tend to breathe orally even at rest. At some level of increased ventilation, individuals who normally breathe through the nose at rest also shift over to oronasal breathing. In regard to ventilation levels at which that shift has been observed to occur, Niinimaa et al. (1980, 1981) reported a switch from nasal to oronasal breathing at a minute volume of 35.3 ± 10.8 (mean ± S. D.) liters, and after the switch to oronasal breathing by persistent nasal breathers (at rest), the nasal portion of Ve decreased to 56 percent of total Ve. In addition to the studies recently published by Niinimaa et al. (1980, 1981), D'Alfonso (1980) has also observed the shift to oronasal breathing in response to increasing ventilation rate and found that subjects who are nasal breathers at rest move to oronasal breathing at a mean Ve of 30 liters per minute. At maximum exercise levels (Ve = 90 liters/minute), subjects breathe, at most, 40% of the total minute volume through the nose.

The results of such studies are extremely important in aiding our understanding of results reviewed here as being derived from controlled human exposure studies of PM and SO_2. Sulfur dioxide, for example, is very soluble in water and, when inhaled nasally, is readily (95 to 99 percent) absorbed on the moist surfaces of the nose and upper respiratory passages (Frank et al., 1973). This may protect individuals breathing nasally at rest from even relatively high levels of SO_2 exposure. At some level of ventilation, however, breathing shifts from nasal to oronasal, thereby increasing the dose of SO_2 reaching the tracheobronchial region of the lung and probably leading to enhanced SO_2 effects at ambient exposure levels below those affecting the same individuals while breathing nasally at rest or at lower activity levels. Forced oral breathing yields less nasopharyngeal absorption than either nasal or oronasal breathing and would be expected to yield a more intense exposure-effect relationship than observed with either nasal or oronasal breathing. The significance of these exposure variables can be discerned clearly when examining the results of available controlled human exposure studies summarized below, especially in regard to SO_2 effects.

Sulfur dioxide has been found to affect a variety of physiological functions. These include sensory processes, subjective perceptions of irritative or painful SO_2 effects, and more objectively measured changes in respiratory function parameters. Although the reliability of subjective reports of perceived effects of SO_2 has been questioned by some, certain statements can be made with confidence concerning SO_2 effects on sensory processes. For example, exposure to 5 ppm of SO_2 results uniformly in the detection of the odor of the gas, while odor detection below that level varies considerably. Other changes (e.g., alterations in electroencephalogram alpha rhythms or in the response of the dark adapted eye to light) have been reported to occur at SO_2-exposure levels as low as 0.20 to 0.23 ppm. The health significance of such "sensory effects" is unclear at this time, but they would appear to be of relatively little concern unless any resulting discomfort or other outcome would markedly alter normal activities of affected subjects.

Of much more concern are cardiovascular or respiratory effects found to be associated with exposure to SO_2. For healthy subjects at rest, in general, such effects have not been consistently observed except at exposure levels above 5 ppm (13.1 mg/m^3). These include, for example, observations by Frank et al. (1962) of marked increases in pulmonary flow resistance (mean = 39%) at 5 ppm (13.1 mg/m^3) and consistent observations by numerous other investigators listed in Table 1-11 of increased airway resistance or other bronchoconstrictive effects with exposures of healthy adult subjects to SO_2 levels of 5 ppm (13.1 mg/m^3) or higher. Only Amdur et al. (1953) have reported observations of significant cardiorespiratory effects in healthy adults at rest following SO_2 exposures below 5 ppm (13.1 mg/m^3), including exposures as low as 1 ppm (2.6 mg/m^3). Other investigators (e.g., Lawther, 1955; Frank et al., 1962) have not observed similar results in attempting to replicate the findings of Amdur et al. (1953) at levels below 5 ppm (13.1 mg/m^3). Numerous accounts could be offered for this apparent discrepancy in reported exposure-effect relationships for bronchoconstriction effects in healthy adults at rest, but no clear resolution of the issue is currently available. Nevertheless, available evidences points to 5.0 ppm (13.1 mg/m^3) as being the most probable lowest observed effect level for induction of bronchoconstrictive effects in healthy adults exposed to SO_2 while at rest.

Probably of more crucial importance are the findings of several investigators suggesting potentiation of SO_2 airway effects in normal subjects as the result of increased oral inhalation of SO_2, due either to forced mouth breathing or increased exercise levels or both. As indicated in Table 1-11, for example, deep breathing of SO_2 at 1 ppm (2.6 mg/m^3) increased specific airway resistance (SR_{aw}) significantly in comparison to breathing air alone (Lawther et al., 1975). Also, Melville (1970) reported greater decreases in specific airway conductance (SG_{aw}) with oral breathing than with nasal breathing at 2.5 ppm (6.6 mg/m^3) SO_2; and Snell and Luchsinger (1969) found significant decreases in maximum expiratory flow ($MEF_{50\%}$) at 1 ppm (2.6 mg/m^3) SO_2 with oral breathing at rest but not at 0.5 ppm (1.3 mg/m^3) SO_2. Similarly, Jaeger et al. (1979) observed no pulmonary effects in resting normal subjects with forced oral breathing at 0.5 ppm (1.3 mg/m^3) SO_2. These studies suggest possible bronchoconstriction effects in healthy adults with oral breathing of 1.0 to 2.5 ppm (2.6 to 6.6 mg/m^3) SO_2,

TABLE 1-11. SUMMARY OF STUDIES ON RESPIRATORY EFFECTS OF SO_2

Concentration SO_2 (ppm)[a]	Duration of exposure (mins)	Number of subjects	Oral (O) or nasal (N) exposure	Rest (R) or exercise (E)*	Effects[b]	Reference
HEALTHY ADULT SUBJECTS AT REST						
10,15,25,50	60	1	-	-	Mucociliary activity decreased at higher conc. (>15 ppm SO_2)	Cralley, 1942
9 - 60	5	10	Mask	R	Airway resistance increased	Nakamura, 1964
5, 10 20	10 10	18 6	O, N O	R R	No changes in pulse rate, resp. rate or tidal vol. (5, 10 ppm). Bronchospasm in two subjects at 10 ppm	Lawther, 1955
1 - 80	10 - 60	8 - 12	Mask, chamber N	R	Bronchoconstriction above 5 ppm	Sim and Pattle, 1957
1 - 45	10	46	Mask	R	Decreased peak flow, decreased expiratory capacity at ≥ 1.6 ppm	Tomono, 1961
1 - 8	10	14	Mask	R	Pulse and respiratory rates increased; tidal volume rate decreased at ≥1.0 ppm	Amdur et al., 1953
1(1-2), 5(4-7), 13(10-16)	10 - 30	11	O*	R	Pulmonary flow resistance for groups increased 39% at 5 ppm and 72% at 13 ppm. At 1 ppm, one subject had 7% increase in flow resistance, another a 23% decrease	Frank et al., 1962
1(1-2), 5(4-6), 15(14-17)	30	12	O*	R	Increases in Rl (pulmonary flow resistance) at ≥5 ppm SO_2	Frank et al., 1964
4 - 6	10	7	O	R	Airway conductance decreased 39%. Blocked by atropine	Nadel et al., 1965
4 - 5	10	5	O*	R	Increased respiratory and inspiratory resistance	Abe, 1967

*Mouthpiece

TABLE 1-11. (continued)

Concentration SO_2 (ppm)	Duration of exposure (mins)	Number of subjects	Oral (O) or nasal (N) exposure	Rest (R) or exercise (E)	Effects	Reference
15, 28	10	8	O,N	R	Pulmonary flow resistance increased less with N breathing	Speizer and Frank, 1966
1.0, 5.0 and 25.0	2-4 hr/d	15	Chamber (N)	R	Significant decreases in expiratory flow and $FEV_{1.0}$ at 25 ppm. Decreased nasal mucus flworate at ≥ 5 ppm. Responses greater after 4 hr than after 2 hr	Andersen et al., 1974
5	270 hr	16 controls 16 exposed	Chamber (N)	R	50% decrease in nasal mucus flowrate but number of colds similar in both groups	Andersen et al., 1977
5	120	9	O	R	No effect on mucus transport	Wolff et al., 1975a
5 - 30 1 3	10 60/DB DB	10 13/12 17	CO_2 stimulus (O)* Chamber N/O O*	R R R	For group as whole (12 subjects) small but significant (14%) increase in SR_{aw} following 25 DB by air alone and 26% increase after 25 DB SO_2 at 1 ppm; but no changes detected after normal quiet breathing of 1-3 ppm SO_2	Lawther, 1975
2.5, 5.0, 10.0	10	15	O*, N	R	Greater percentage decrease in in SG_{aw} with O breathing at all concentrations	Melville, 1970
0.5, 1.0, 5.0	15	9 5	O* O, N*	R R	Decreases in $MEF_{50\%\ VC}$ for group were sig. at 1 and 5 ppm SO_2; at 5 ppm, decreases for N not sig. different from O breathing	Snell and Luchsinger, 1969
1.1 - 3.6	30	10	O*	R	Deep breathing produced no effects	Burton et al., 1969
0.50	180	40	Chamber (O) Nose clips	R	No pulmonary effects seen	Jaeger et al., 1979

*Mouthpiece
DB = deep breaths

TABLE 1-11. (continued)

Concentration SO_2 (ppm)	Duration of exposure (mins)	Number of subjects	Oral (O) or nasal (N) exposure	Rest (R) or exercise (E)	Effects	Reference
EXERCISING HEALTHY ADULTS						
5.0	120	10	O	E	Increased tracheobronchial clearance	Wolff et al., 1975b
5.0	120	11	Chamber	E	Insignificant changes in airway resistance and arterial PO_2	von Neiding et al., 1979
5.0	120	10	Chamber (O)	E	MMFR decreased 8.5%; increased tracheobronchial clearance	Newhouse et al., 1978
5.0	3	10	O	R	Light exercise potentiates effect of SO_2; $MEF_{40\%}$ decreased at 3 ppm and above	Kreisman et al., 1976
3.0	3	8+9	O	R + E		
1.0	3	10+8	O	R + E		
0.5	3	5	O	R		
0.75	120	4	Chamber	E	Decrease in MMFR, FVC, $FEV_{1.0}$ (~8-10%) and 20% in $MEFR_{50\%}$	Bates and Hazucha, 1973
0.75	120	15 controls 16 exposed	Chamber	E	Significantly elevated Raw and trend toward decreased FEF_{50} and FEV/FVC after SO_2 exposure during heavy exercise	Stacy et al., 1981
0.50	120	24	Chamber	E	No pulmonary effects seen with 0.50 ppm SO_2 + 0.5 ppm NO_2	Linn et al., 1980
0.40	120	9	Chamber	E	No pulmonary effects	Horvath and Folinsbee, 1977; Bedi et al., 1979
0.40	120	11	Chamber	E	No pulmonary effects seen with 0.4 ppm SO_2 alone	Bedi et al., 1981
0.37	120	8	Chamber	E	No pulmonary effects	Bates and Hazucha, 1973; Hazucha and Bates, 1975
0.37	120	4-12	Chamber	E	No pulmonary effects	Bell et al., 1977.
RESPIRATORY DISEASE SUBJECTS						
0.3, 1.0 and 3.0	96 - 120 hr	12 (normal) 7 (COPD)	Chamber	R	No difference in response between groups. Slight decrease in pulmonary compliance but of questionable significance	Weir and Bromberg, 1972
7.7	6 d	32 normals	Chamber (N)	R	No significant changes in airway resistance or other effects in health subjects or patients	Reichel, 1972
0.3-4	6-7 d	27 subjects w/obstrutive resp. disease	Chamber (N)	R		

TABLE 1-11. (continued)

Concentration SO_2 (ppm)	Duration of exposure (mins)	Number of subjects	Oral (O) or nasal (N) exposure	Rest (R) or exercise (E)	Effects	Reference
ASTHMATIC SUBJECTS						
1, 3, 5	10	7 normals 7 atopics 7 asthmatics	O[*]	R	SR_{aw} increased significantly at all conc for asthmatics; only at 5 ppm for normals and atopic subjects. Some asthmatics exhibited marked dyspnea requiring bronchodilation therapy.	Sheppard et al., 1980
1.0 0.1, 0.25, 0.5	5 10	6 asthmatics 7 asthmatics	O[*]	E	SR_{aw} significantly increased in the asthmatic group at 0.5 and 0.25 ppm SO_2 and at 0.1 ppm in the two most responsive subjects. At 0.5 ppm three asthmatics developed wheezing and shortness of breath.	Sheppard et al., 1981
0.50	180	40 (asthmatics)	Chamber (O) Nose clips	R	MMFR significantly decreased 2.7%; recovery within 30 min.	Jaeger et al., 1979
0.5	10	5 asthmatics	O[*]	E	Specific airway resistance (SR_{aw}) increases were observed over exercise baseline rates for 80% of the subjects.	Linn et al., 1982
0.25, 0.5	60	24 asthmatics	Chamber	E	No statistically significant changes in forced vital capacity (FVC) or specific airway resistance (SR_{aw})	Linn et al., 1982
0.30	120	19 (asthmatics)	Chamber	E	No pulmonary effects seen with 0.3 ppm SO_2 and 0.5 ppm NO_2 exposure compared to exercise basline	Linn et al., 1980

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[a] 0.1 ppm SO_2 ≅ 262 μg/m³ 1.0 ppm ≅ 2620 μg/m³ 10 ppm ≅ 26,200 μg/m³
0.5 ppm SO_2 ≅ 1310 μg/m³ 5.0 ppm ≅ 13,100 μg/m³ 50 ppm ≅ 131,000 μg/m³

[b] Significant increase or decreases noted here refer to "statistically significant" effects, independent of whether the observed effects are "medically significant" or not.

[c] Chronic obstructed pulmonary disease.

[*] Mouthpiece DB = deep breaths

raising the possibility of such effects being seen at similar concentrations in healthy adults exercising at sufficient workloads to induce a shift to oronasal breathing.

Examining the effects of exercise, Kreisman et al. (1976) found that light exercise potentiated the effect of SO_2, with $MEF_{40\%}$ being significantly decreased with exercise during oral exposure of normal subjects to 3 ppm (7.9 mg/m^3) SO_2 or above. Another study, by Bates and Hazucha (1973), reported a 20 percent (but not statistically significant) decrease in MEFR with 0.75 ppm (2.0 mg/m^3) exposure of exercising adults in an open chamber; and Stacy et al. (1981) reported slight but statistically significant SR_{aw} increases in healthy adults exposed to 0.75 ppm (2.0 mg/m^3) SO_2 while exercising in a controlled exposure chamber. These effects were the only significant ones found among numerous pulmonary function test results even under rather extreme exercise conditions employed in the Stacy et al. (1981) study. These results (Bates and Hazucha, 1973; Stacy et al. 1981) therefore provide only very weak evidence for effects in exercising healthy adults at SO_2 levels <1.0 ppm (2.6 mg/m^3). In other studies, no pulmonary effects were observed with chamber exposures of exercising healthy adults at SO_2 exposure levels of 0.50, 0.40, or 0.37 ppm (1.31, 1.05, or 0.97 mg/m^3) (Horvath and Folinsbee, 1977; Bedi et al., 1979; Bates and Hazucha, 1973; Hazucha and Bates, 1975; Bell et al., 1977; Linn et al., 1980; Bedi et al., 1981). The weight of available evidence, therefore, appears to indicate that induction of pulmonary mechanical function effects may occur with exposure to concentrations of 1 to 3 ppm (2.6 to 7.9 mg/m^3) SO_2 or higher in exercising healthy adults but not at ≤0.50 ppm (1.31 mg/m^3) SO_2 even with exercise or forced oral breathing.

In attempting to define populations at special risk for SO_2 effects, Weir and Bromberg (1972) and Reichel (1972) exposed patients with obstructive pulmonary disease to SO_2 levels across the range of 0.3 to 4.0 ppm (0.8 to 10.5 mg/m^3) and observed no statistically significant increase in airway resistance or other pulmonary function effects. The exposures were carried out while the subjects were at rest in a controlled exposure chamber, but no assessment was conducted regarding possible enhanced effects of increased oral inhalation due to exercise or forced mouth breathing. Thus, although no evidence was obtained for increased susceptibility of these patients at rest, possibly enhanced vulnerability to SO_2 effects of such subjects at elevated activity levels cannot be ruled out based on the reported results.

A clearer picture of probable enhanced susceptibility or special risk for SO_2-pulmonary function effects appears to be emerging now in regard to asthmatic subjects. For example, Jaeger et al. (1979) reported observing small, statistically significant (mean = 2.7%) decreases in MMFR levels (which recovered in 30 minutes) following forced oral exposure (by use of nose clips) to 0.5 ppm (1.3 mg/m^3) SO_2 of 40 asthmatic subjects at rest in a controlled exposure chamber. Two subjects experienced delayed effects requiring medication that may have been due to the SO_2 exposures. (Other uncontrolled factors, however, cannot be ruled out as possibly having caused the delayed symptoms.) While the small pulmonary function decrements observed by Jaeger et al. (1979) may be physiologically insignificant per se, they are suggestive of possible SO_2 effects occurring in asthmatic individuals at SO_2 levels below those affecting nonsensitive healthy adults.

Consistent with this possibility, Sheppard et al. (1980) observed statistically significant SR_{aw} increases in subjects with clinically defined mild asthma exposed to 1, 3, or 5 ppm (2.6, 7.7 or 13.1 mg/m^3) SO_2 via mouthpieces while at rest; however, significant SR_{aw} increases in normal and atopic subjects occurred only at 5 ppm (13.1 mg/m^3). In further studies, Sheppard et al. (1981) observed statistically significant increases in SR_{aw} with oral exposure of asthmatic subjects to 0.25 and 0.5 ppm (0.7 and 1.3 mg/m^3) SO_2 via forced mouth breathing while exercising at a moderately elevated level ($Ve \cong 30$ liters). The two most responsive subjects of six tested experienced increased SR_{aw} with oral exposure to levels as low as 0.10 ppm (260 mg/m^3) SO_2. At 0.5 ppm three of the subjects experienced wheezing and shortness of breath, and at 1.0 ppm all six subjects experienced such symptoms. Sheppard et al. (1980) also employed pharmacologic tests, which indicated that the very rapid-onset bronchoconstriction effects seen in the asthmatic subjects are under parasympathetic neural control, as was earlier demonstrated (Nadel et al., 1965) to be the case for normal subjects experiencing bronchoconstriction in response to exposure to SO_2 at a higher level (i.e., 5 ppm) while at rest.

The Sheppard et al. (1980, 1981) results appear to demonstrate that some asthmatic subjects may be approximately an order of magnitude more sensitive to SO_2 exposure than normal, nonsensitive healthy adults. That is, whereas nonsensitive healthy adults display increased bronchoconstriction at 5 to 10 ppm while at rest and at levels possibly as low as 1 ppm with oral or oronasal breathing, persons with clinically defined mild asthma appear to be sensitive (as a group) down to 0.25 ppm SO_2 and the most sensitive (as individuals) down to 0.1 ppm under moderate exercise ($Ve \cong 30$ liters/minute) conditions. Most importantly, with brief 10-minute exposures to SO_2 concentrations encountered in U.S. cities (0.1 to 0.5 ppm), Sheppard et al. (1981) demonstrated that moderate exercise increased the bronchoconstriction produced by SO_2 in subjects with mild asthma. These results were qualitatively confirmed by Linn et al. (1982) using techniques similar to those employed by Sheppard et al. (1981). In a pilot study by Linn et al. (1982), five asthmatic subjects were exposed, via mouthpiece, to 0.5 ppm SO_2 for a period of 10 minutes while exercising (at a rate equivalent to ~400 kg-m/ min), and 4 of the 5 showed increased SR_{aw} in response to the SO_2 exposure. Similar results using oronasal 0.5 ppm SO_2 exposure via a face mask have been recently described (see Addendum following Chapter 1 in this volume). However, caution should be employed in regard to any attempted extrapolation of these observed quantitative exposure-effect relationships to what might be expected under ambient conditions. Additional research results from studies using open chamber oronasal breathing conditions more analogous to those encountered in daily activities have also recently been described by Linn et al. (1982). In this large-scale chamber study employing 24 asthmatic subjects, no statistically significant pulmonary function decrements were found with 0.5 ppm SO_2 exposures for 1 hour under intermittent exercising conditions. These negative results are in contrast to the findings of Sheppard et al. (1981) and Linn et al. (1982) obtained with 0.5 ppm SO_2 exposure via mouthpiece while exercising. These differences may be due to the delivery of a higher proportion of inhaled SO_2 to the tracheobronchial and lung regions with mouthpiece exposure or to individual variations in bronchial reactivity to SO_2 among subjects used in the different studies.

The health significance of pulmonary function changes and associated symptomatic effects demonstrated to occur in response to SO_2 by the above human exposure studies is an important issue for present air quality criteria development purposes. In contrast to the sensory effects of SO_2 earlier described as probably being of little health significance, much more concern is generally accorded to the potential health effects of pulmonary function changes (such as increased bronchoconstriction) and associated symptomatic effects (such as coughing, wheezing, and dyspnea or shortness of breath) observed with human exposures to SO_2, especially in sensitive population groups such as those having asthma. Temporary, small decrements in pulmonary airway functions observed in some of the above studies for nonsensitive healthy adults at SO_2 concentrations of greater than 1 to 5 ppm are generally of less concern in terms of their implications regarding the potential health impact of ambient air SO_2 exposures than are the pulmonary function and symptomatic effects observed in mildly asthmatic persons at similar (1 to 5 ppm) or lower (<1 ppm) concentrations of SO_2. Probably of most concern are marked increases (>10 percent) in airway resistance and symptomatic effects (wheezing, dyspnea) observed by Sheppard et al. (1981) in a group of mildly asthmatic subjects with oral exposure via mouthpiece to 0.5 ppm (1.3 mg/m^3) SO_2 during exercise, although the level of SO_2 exposure at which such effects might occur under ambient conditions cannote be precisely stated at this time. A recent article (Fischl et al., 1981) and accompanying editorial (Franklin, 1981) in the medical literature discuss the inclusion of indices of airway obstruction and symptoms such as wheezing and dyspnea among factors to be considered in attempting to predict the need for hospitalization of asthma patients following initial emergency room treatment (e.g., bronchodilator therapy, etc.) for asthma attacks.

Particulate matter, especially hygroscopic salts, has been shown to be potentially important in enhancing the pulmonary function effects of SO_2 exposure. Airway resistance increased more after combined exposure to SO_2 and sodium chloride in several studies, although others have failed to demonstrate the same effect. This difference in response to the SO_2-NaCl aerosol mixtures may be due principally to the relative humidity at the time of the exposure. McJilton et al. (1973) have demonstrated that changes in pulmonary mechanical function were seen in guinea pigs only when the SO_2-NaCl mixture was administered at high relative humidity (RH >80%). The effect is ascribed to absorption of the highly soluble SO_2 into the droplet before inhalation, whereas at RH <40% the aerosol was a crystal. Significant reduction in $MEF_{50\%VC}$ was observed for the group mean after oral exposure to a combination of saline aerosol and 5 ppm (13.3 mg/m^3) SO_2; however, no effects were observed at SO_2 levels of 0.5 and 1.0 ppm (1.3 and 2.6 mg/m^3) (Snell and Luchsinger, 1969). The validity of this study has been questioned because of the lack of an air sham control group and also based on the methodology used to measure $MEF_{50\%VC}$. More recently, studies have been reported showing pulmonary function changes in extrinsic asthmatic subjects both at rest (Koenig et al., 1980) and during exercise (Koenig et al., 1981) with exposure to 2.62 mg/m^3 (1 ppm) SO_2 and 1 mg/m^3 NaCl. Statistically significant decreases in certain measures of maximum expiratory flow ($V_{max50\%}$ and $V_{max75\%}$) were observed both at rest and during exercise for asthmatic subjects but not for all normal subjects. Although NaCl alone produced no such effects, the lack of a group exposed to SO_2 alone and the difference in the number of subjects used with NaCl alone or in combination with SO_2 make interpretation difficult.

In contrast to the apparent enhancement of SO_2-induced pulmonary airway effects by combined exposure with certain particulate matter aerosols, there is less evidence that synergistic interactions between SO_2 and other gaseous pollutants, such as ozone or nitrogen dioxide, produce greater-than-additive effects on pulmonary mechanical functions. None of the controlled human exposure studies reviewed in Chapter 13 convincingly demonstrated such synergistic effects.

Evidence from controlled human exposure studies regarding SO_2 effects on respiratory defense mechanisms, such as mucus clearance processes, is highly limited at present. For healthy adults exposed to SO_2 while at rest, nasal mucus flowrate appeared to decrease markedly (by 50 percent) at 5.0 ppm SO_2 (Andersen et al., 1977), but tracheobronchial mucociliary clearance appeared to be unaffected by SO_2 exposure at the same level while at rest (Wolff et al., 1975a). These observed differences may be due to the much greater dose of SO_2 delivered to nasal passages than to tracheobronchial regions by nasal breathing at rest. Oral exposure of healthy adults to 5.0 ppm (13.1 mg/m^3) SO_2 during exercise (which notably increases tracheobronchial deposition of SO_2), however, was observed to increase tracheobronchial clearance rates in two studies (Wolff et al., 1975b; Newhouse et al., 1978). No studies, to date, have investigated whether or not repeated exposures to 5.0 ppm SO_2 would continue to induce increased nasal or tracheobronchial clearance or, possibly, cause eventual slowing of mucus clearance. (Note that one early study by Cralley [1942] reported decreased mucociliary activity in a healthy adult exposed to high [>15 ppm] SO_2 concentrations while at rest.) Nor have any controlled exposure studies investigated the effects of SO_2 exposure on mucus clearance activities in asthmatic or other potentially sensitive human population groups, such as individuals with chronic obstructive pulmonary diseases. Thus, while SO_2 effects on nasal and tracheobronchial mucus clearance processes cannot be said to have been demonstrated to occur in sensitive population groups at exposure levels below those affecting healthy adults, such a possibility cannot be ruled out at this time.

In addition to SO_2 being absorbed by hygroscopic particles, whereby its effects may be potentiated, sulfur dioxide is also transformed during transport into sulfur trioxide which in turn in combination with moisture forms sulfuric acid. The latter may exist as a sulfuric acid droplet or can be converted to sulfates in the presence of ammonia, which is found in the ambient air and in expired human breath.

Sulfuric acid and other sulfates have been found to affect both sensory and respiratory function in study subjects. The odor threshold for sulfuric acid has been estimated to be at 0.75 mg/m^3 based on one study and at 3.0 mg/m^3 based on another.

Respiratory effects from exposure to sulfuric acid mist (0.35 to 0.5 mg/m^3) have been reported to include increased respiratory rate and decreased maximal inspiratory and expiratory flowrates and tidal volume (Amdur et al., 1952). However, several other studies of pulmonary function in nonsensitive healthy, adult subjects (Newhouse et al., 1978; Sackner et al., 1978; Kleinman et al., 1978; Avol et al., 1979; Leikauf et al., 1981; Kerr et al., 1981; Horvath et al., 1981) indicated that pulmonary mechanical function was little affected when subjects were exposed at 0.1 to 1.0 mg/m^3 sulfuric acid for 10 to 120 minutes, although in one study (Utell et al., 1981) the bronchoconstrictive action of carbachol was potentiated by the sulfuric acid and sulfate aerosol, more or less in relation to their acidity.

In regard to mucociliary clearance effects, tracheobronchial clearance was significantly increased at 100 µg/m^3 H_2SO_4, was not significantly altered at 300 µg/m^3, but was significantly decreased at 1000 µg/m^3 (Leikauf et al., 1981). Although transiently depressed following a single 60-minute exposure, the decreased clearance rates seen at 1000 µg/m^3 raise the possibility of more persistent or chronic depression of tracheobronchial clearance after repeated exposures to the same concentrations of sulfuric acid. The possible occurrence of such an effect in humans would be consistent with observations of persistently slowed clearance for several months following repeated exposures of donkeys to comparable H_2SO_4 concentrations (Schlesinger et al., 1978, 1979).

In studies with asthmatic subjects, no changes in airway function have been demonstrated after exposure to sulfuric acid and sulfate salts at concentrations less than 1000 µg/m^3. However, at concentrations higher than 1000 µg/m^3, reductions in specific airway conductance (SG_{aw}) and forced expiratory volume ($FEV_{1.0}$) have been observed after sulfuric acid and ammonium bisulfate exposures, as reported by Utell et al. (1981). No studies, on the other hand, have as yet evaluated the effects of sulfuric acid or other sulfate salt aerosols on nasal or trancheobronchial mucus clearance functions.

Water-soluble sulfates have been the most frequent ingredients of experimental aerosol exposure atmospheres because ambient sulfate levels were earlier reported as likely being epidemiologically associated with morbidity. However, in addition to sulfuric acid and sulfates, other nonsulfur particulate matter species exist in the ambient air. These include polycyclic organic matter (POM), lead, arsenic, selenium, ammonium salts, and carbon as dust. Although controlled human exposure to some of these inherently toxic compounds is forbidden for obvious reasons, several investigators have conducted clinical studies using carbon and other inert particles.

The relatively sparse results involving insoluble and other nonsulfur aerosols under controlled human exposure conditions preclude drawing conclusions regarding quantitative exposure-effect or dose-response relationships for the particulate chemical species studied. This is due to the fact that extremely high aerosol concentrations were typically employed in such studies. Nor can any clear conclusions be drawn, based on the available controlled human exposure data, in regard to size ranges of insoluble and other nonsulfur aerosols that may be associated with the induction of significant respiratory system effects at concentrations commonly found in the ambient air (although most of the controlled exposure studies generally appear to have employed either fine particles of <2.5 μm diameter or inhalable particles of <10-15 μm diameter). However, the effects in polydispersed aerosol studies cannot be ascribed to fine particles alone. Only studies by McDermott (1962), Anderson et al. (1979), and Toyoma and Nakamura (1964) have explicitly studied the effects of larger particles but at highly elevated levels of insoluble particulate matter not usually associated with ambient conditions.

1.14. EPIDEMIOLOGICAL STUDIES ON HEALTH EFFECTS OF PARTICULATE MATTER AND SULFUR OXIDES

Chapter 14 evaluates epidemiological literature concerning health effects associated with ambient air exposures to particulate matter and sulfur oxides. The main focus of the chapter is on: (1) qualitative characterization of human health effects associated with exposure to airborne SO_2, related particulate sulfur compounds, and other PM; (2) quantitative delineation of exposure-effect and exposure-response relationships for induction of such effects; and (3) identification of population groups at special risk for experiencing the effects at ambient exposure levels. The epidemiological data discussed both complement and extend information presented as part of analyses in other health-related chapters (11, 12, and 13) of the document. Epidemiological studies offer several advantages beyond those of animal toxicology or controlled human exposure studies. Health effects of both short- and long-term pollutant exposures (including complex mixtures of pollutants) can be studied and sensitive members of populations at special risk for particular effects at ambient air concentrations identified. Also, epidemiological evaluations allow for investigation of both acute and chronic disease effects and associated human mortality. Epidemiological studies, then, together with controlled animal and human exposure studies, can contribute to a more complete understanding of the health effects of PM and SO_x, especially in helping to delineate human health effects occurring under ambient exposure conditions. Despite these advantages, however, important limitations exist regarding the conduct, analysis, interpretation, and use of available epidemiological studies on the health impact of PM and SO_2. Such limitations, summarized next, are discussed in more detail in Section 14.1.1 of Chapter 14 and must be taken into account in any evaluation of epidemiological studies on PM and SO_x.

1.14.1 Methodological Considerations

Epidemiological studies employed to generate information for human risk assessment purposes typically focus on the following: (1) defining exposure conditions; (2) identifying health effects; (3) relating exposures to effects; and (4) estimating overall risk of particular health effects occurring among specific population groups under ambient exposure conditions.

One important limitation of most epidemiological studies reviewed in Chapter 14 is less-than-optimum characterization of community air quality parameters used to estimate exposures of population groups to atmospheric concentrations of PM and SO_2. Such characterization of air quality generally involved relatively crude estimates of levels of pollutants present, often allowing for only limited qualitative statements to be made regarding exposure conditions (e.g., whether a given site or time period had higher or lower atmospheric levels of PM or SO_2 than some other site or time period). Only rarely were measurement methods used that provided reasonably precise determinations of ambient air concentrations of pollutants, which were sufficient to permit quantification of approximate PM or SO_2 levels associated with observed health effects. Even when reasonable quantification of community air quality parameters was achieved, however, the use of such data in estimating actual population exposures was typically further constrained by factors such as siting of air sampling devices in relation to study populations, frequency and duration of sampling periods, activity patterns of study population members, and contributions of indoor air pollution to overall exposures of study groups. These limitations arise in part from the fact that most presently-available epidemiological studies utilized air monitoring data from sampling networks originally established for purposes other than health-related research and, therefore, not optimally designed to provide aerometric data of the type or quality needed for precise epidemiological assessment of health effects related to PM and SO_2. Therefore, the aerometric data reported should be viewed as yielding, at best, only approximate estimates of actual study population exposures.

Inadequate characterization of health effects associated with PM and SO_2 exposure conditions represents another major problem with many of the epidemiological studies evaluated. Various health endpoint measurements (mortality, morbidity, and indirect measures of morbidity) were employed in such studies and each has advantages and disadvantages. Some involved direct observations of signs and symptoms of disease states or objective indicators thought to be associated with the occurrence of illnesses, e.g., patient visits to hospitals or clinics or absenteeism from school or work. Direct quantification of health effects also included measurement of biochemical or physiological changes in study populations, as in recording of pulmonary function changes by spirometric methods. Indirect measures or indices of health effects were also used, e.g., by gathering information on frequency and duration of respiratory illnesses by telephone interviews, written questionnaires, or self-reported entries in diaries. The validity of such indirect measurements of health effects, however, is highly dependent on the ability and motivation of respondents to recall and report accurately past or present health-related events; this can be influenced by numerous extraneous factors such as age, cultural and educational background, instructions from experimenters, sequencing of questions, and interviewer variability or bias. Confidence in results obtained by either direct or indirect measurement methods is enhanced if potentially interfering or biasing factors are appropriately controlled for and if results are validated against corroborating evidence, but very few of the available epidemiological studies on PM and SO_2 effects adequately addressed such methodological issues.

Adequately relating observed health effects to specific parameters of ambient exposure conditions is another objective not often achieved by the epidemiological studies reviewed, such that few allow for confident qualitative or quantitative characterization of PM or SO_2 exposure-health effect relationships. For example, competing risks such as cigarette smoking and occupational exposures may contribute to observed health effects results and must usually be taken into account in order for confidence to be placed in reported air pollution-health effects relationships; however, many studies on PM or SO_2 effects did not adequately control for such factors. Similarly, possible effects of other covarying or confounding factors (e.g., socioeconomic status, race, and meteorological parameters) were not always adequately evaluated. Also, further complicating evaluation of the epidemiological data is the fact that exposure parameters are not subject to experimenter control; thus, ambient levels of a given pollutant often varied widely over the course of most studies, making it extremely difficult to determine whether mean concentrations, peak concentrations, rapid fluctuations in levels, or other air quality factors were most important as determinants of reported health effects. Significant covariation between concentrations of PM, SO_2, and other pollutants also often made it difficult to distinguish among their relative contributions to observed health effects.

Estimation of overall risk by means of epidemiological studies requires still further steps beyond delineation of exposure-effect relationships that define exposure conditions (levels, durations, etc.) associated with induction of specific health effects. That is, risk estimation also requires: (1) identification of particular population groups likely to manifest health effects under exposure conditions of concern; and (2) ideally, determination of numbers or percentages of such individuals (responders) likely to be affected at various exposure or dose levels. Delineation of the former, i.e., identification of population groups at special risk at comparatively low exposure levels of SO_2 and PM, has only started to be accomplished via presently available epidemiological studies. Also, epidemiological delineation of quantitative dose-response (or, more correctly, exposure-response) relationships, defining percentages of population groups likely to manifest a given health effect at various levels or durations of exposure to PM and SO_2, is largely lacking at this time.

Another limitation of the epidemiological information concerns its usefulness in demonstrating cause-effect relationships versus merely establishing associations (which may be non-causal in nature) between PM or SO_2 and various health effects. Interpretation of epidemiological data as an aid in inferring causal relationships has been addressed by previous expert committees or deliberative bodies faced with evaluation of controversial biomedical issues (U.S. Surgeon General's Advisory Committee on Smoking and Health, 1964; U.S. Senate Committee on Public Works, Subcommittee on Air and Water Pollution, 1968). Among criteria selected by each group were: (1) the strength of the association; (2) the consistency of the association, as evidenced by its repeated observation by different persons, in different places, circumstances, and time; (3) the specificity of the association; (4) the temporal relationship of the association; (5) the coherence of the association in being consistent with other known facts; (6) the existence of a biological gradient, or dose-reponse curve, as revealed by the association; and (7) the biological plausibility of the association. In discussing such criteria, Hill (1965) further noted that strong support for likely causality suggested by an association may be derived from experimental evidence, where manipulation of the presumed causative agent (its presence or absence, variability in intensity, etc.) also affects the frequency or intensity of the associated effects. Importantly, Hill (1965) and the above committees emphasized, regardless of the specific set of criteria selected by each, that no one criterion was definitive by itself nor was it necessary that all be fulfilled in order to support a determination of causality. Also, Hill and the committes noted that statistical methods cannot establish proof of a causal relationship in an association nor does lack of "statistical significance" of an association necessarily negate the possibility of a causal relationship. That is, as stated by the U.S. Surgeon General's Advisory Committee on Smoking and Health (1964): "The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability." All of the above points are important to consider in arriving at conclusions regarding the meaning and implications of the epidemiological data evaluated in the present document.

Taking into account the above methodological limitations, the following set of guidelines are stated in Chapter 14 and were used to judge the relative scientific quality of epidemiological studies and their findings reviewed there:

1. Was the quality of the aerometric data sufficient to allow for meaningful characterization of geographic or temporal differences in study population pollutant exposures in the range(s) of pollutant concentrations evaluated?

2. Were the study populations well-defined and adequately selected so as to allow for meaningful comparisons between study groups or meaningful temporal analyses of health effects results?

3. Were the health endpoint measurements meaningful and reliable, including clear definition of diagnostic criteria and consistency in obtaining dependent variable measurements?

4. Were the statistical analyses appropriate and properly performed and interpreted, including accurate data handling and transfer during analyses?

5. Were potentially confounding or covarying factors adequately controlled or taken into account in the study design and statistical analyses?

6. Are the reported findings internally consistent, biologically plausible, and coherent in terms of consistency with other known facts?

Few, if any, of the epidemiological studies reviewed dealt with all of the above points in a completely ideal fashion; nevertheless, these guidelines provided benchmarks for judging the relative quality of various studies and for selecting the best for detailed discussion in Chapter 14.

Detailed critical analysis of all epidemiological studies on health effects of PM and SO_2 represents an undertaking beyond the scope of the present document. Of most importance for present purposes are those studies which provide useful quantitative information on exposure-effect or dose-response relationships for health effects associated with ambient air levels of PM and SO_2 likely to be encountered in the United States during the next 5 years. Accordingly, the following criteria were employed in selecting studies for detailed discussion in the text of Chapter 14:

1. Concentrations of both PM and SO_2 were reported, allowing for potential evaluation of their separate or combined effects.
2. Study results provided information on quantitative relationships between health effects and ambient air PM and SO_2 levels of current concern (i.e., generally ≤ 1000 $\mu g/m^3$).
3. Important methodological considerations were adequately addressed, especially (a) in controlling for likely potentially confounding factors and (b) in carrying out data collection, analysis, and interpretation so as to minimize errors or potential biases which could be reasonably expected to affect the results.
4. The study results have been reported in the open literature or are in press, typically after having undergone peer review.

In addition, some studies not meeting all of the above criteria are briefly discussed in Chapter 14 as appropriate to help elucidate particular points concerning the health effects of PM and/or SO_2. Other studies found to be of very limited usefulness for present criteria development purposes are noted in Appendix 14A of Chapter 14, along with annotated comments on methodological or other factors that limit their usefulness for present purposes.

The extensive epidemiological literature on the effects of occupational exposures to PM and SO_2 presently is not reviewed in Chapter 14 for several reasons:

1. Such literature generally deals with effects of exposures to SO_2 or PM chemical species at levels many times higher than those encountered in the ambient air by the general population.
2. Populations exposed occupationally mainly include healthy adults, self-selected to some extent in terms of being better able to tolerate exposures to SO_2 or PM substances than more susceptible workers seeking alternative employment or other groups often at special risk among the general public (e.g., the old, the chronically ill, young children, and asthmatic individuals).
3. Extrapolation of observed occupational exposure-health effects relationships (or lack thereof) to the general public could, therefore, be potentially misleading in demonstrating health effects among healthy workers at higher exposure levels than would affect susceptible special risk groups in the general population.

The occupational literature does, however, demonstrate links between acute high level or chronic lower level exposures to SO_2 or many different PM chemical species and a variety of health effects, including: pulmonary function changes, respiratory tract diseases, morphological damage to the respiratory system, and respiratory tract cancers. The reader is referred to National Institute of Occupational Safety and Health (NIOSH) criteria documents and other assessments listed in Appendix 14B of Chapter 14 for information on health effects associated with occupational airborne exposures to SO_2 and various PM species.

1.14.2 Air Quality Measurements

Of key importance for evaluation of epidemiological studies reviewed in Chapter 14 is a clear understanding of physical and chemical properties of PM and SO_2 indexed by measurement methods (and associated limitations) used to collect aerometric data employed in those studies. The most crucial points discussed in Chapters 3 and 14 on the subject are summarized here.

Three main measurement methods or variations were used to generate SO_2 data cited in the epidemiological studies reviewed: (1) sulfation rate; (2) hydrogen peroxide; and (3) the West-Gaeke (pararosaniline) methods. As noted earlier (Section 1.3), sulfation rate (lead dioxide) methods are not SO_2-specific, and atmospheric concentrations of SO_2 or other sulfur compounds cannot be accurately extrapolated from the results. However, lead dioxide gauges, widely used in Britain prior to 1960, provided aerometric data reported in some British epidemiological studies, and sulfation rate methods were also used in some American studies. Use of a better method, the hydrogen peroxide method, was expanded in Britain during the 1950s, usually in tandem with apparatus for PM (smoke) monitoring, and the method was adopted in the early 1960s as the standard SO_2 method for the United Kingdom National Survey of Air Pollution and, as an OECD-recommended method, elsewhere in Europe. The method can yield reasonably accurate estimates of atmospheric SO_2 levels expressed in $\mu g/m^3$; but results can be affected by factors such as temperature, atmospheric ammonia, and titration errors. Unfortunately, little quality assurance information exists on sources and magnitudes of errors encountered in the use of the method to obtain SO_2 data reported in specific British or European epidemiological studies, making it difficult to assess the accuracy or precision of reported SO_2 values. The West-Gaeke (pararosaniline) method was more widely used in the United States and is specific for SO_2 if properly implemented to minimize interference by nitrogen or metal oxides; but results can also be affected by factors such as temperature and mishandling of reagents. Again, unfortunately, only very limited quality assurance information (see Appendix 14B of Chapter 14) has been reported for some American SO_2 measurements by the West-Gaeke method but is otherwise generally lacking by which to evaluate the quality of SO_2 data reported in most published American epidemiological studies.

Measurement approaches for suspended sulfates and sulfuric acid, used mainly in the United States, include turbidimetric and methylthymol blue methods, which usually involve collection of samples on sulfate-free glass fiber filters by high-volume PM samplers. However, as discussed in Section 1.3, such methods usually do not differentiate between sulfates and sulfuric acid, and secondary formation of such products from SO_2 in air drawn through the filter can affect estimation of atmospheric sulfate levels. Essentially none of the available epidemiological studies using sulfate aerometric data derived from these measurement methods adequately controlled for such artifact formation; and few other studies have employed more recently developed better sulfate measurement methods.

To be of maximum value, epidemiological studies on PM effects must utilize aerometric methods that provide meaningful data regarding not only the mass but also the size and chemical composition of particles present. In actual practice, most epidemiological studies on PM effects relied on air quality data from air monitoring instruments of questionable sampling accuracy and not specifically designed for health-related research. The resulting data thus typically provided only limited information regarding mass, size, or chemical properties of the PM sampled.

Three measurement approaches were mainly used to obtain PM data cited in the epidemiological studies reviewed: (1) the British Smokeshade (BS) light reflectance method or variations used in Britain and Europe; (2) the American Society for Testing and Materials (ASTM) filter-soiling light transmittance method or AISI variation used in the United States; and (3) the high-volume sampling method widely employed in the United States. As noted in Section 1.3, the BS method in routine use typically employed standard monitoring equipment with a D_{50} cutpoint of ~4.5 µm. Also, as noted earlier, the BS method neither directly measures the mass nor determines chemical composition of collected particles. Rather, reflectance of light from the stain is measured and depends both on density of the stain and optical properties of the collected materials, of which smoke particles composed of elemental carbon typically make the greatest contribution. Because highly variable proportions of carbon and non-carbon PM exist from site to site or from time to time at the same site, the same BS reflectance can be associated with different concentrations of particles. Site-specific calibrations of reflectance readings against gravimetric mass measurements are therefore necessary to obtain approximate estimates of airborne PM concentrations by the BS method. Unfortunately, such site-specific calibration of BS reflectance readings against gravimetric mass measurements was carried out only once in London during the 1950s. Later, in the early 1960s, additional calibrations were carried out, e.g., some site-specific BS mass calibration curves were determined for urban areas in Britain and Europe for British National Survey and OECD work, respectively. Such curves were interrelated or normalized to define two "standard" curves: (1) a British standard smoke curve defining relationships between PM mass and BS reflectance readings for London's atmosphere in 1963, which was used to yield BS concentration estimates (in $\mu g/m^3$) reported in many published British epidemiological studies; and (2) an OECD international standard smoke curve, against which smoke reflectance measurements made elsewhere in Europe were compared to yield smoke concentration estimates (in $\mu g/m^3$) reported in European studies on PM effects. Of crucial importance in assessing such studies is the fact that the actual PM mass or smoke concentration at a particular site may differ markedly (e.g., by factors of two or more) from the corresponding mass or concentration (in μg/m^3) associated with a given reflectance reading on either of the two standard curves[*]; therefore, great care must be applied in interpreting what any reported BS value in μg/m^3 means at all. Further complicating interpretation of smoke data used in most epidemiological studies is the lack of specific quality assurance information for cited aerometric measurements.

The ASTM or AISI light transmittance method is similar in approach to the BS method, having a D_{50} cutpoint of ~5 μm and accumulating PM as a stain on filter paper. Thus, coefficient of haze (CoH) readings (like BS readings) roughly index the soiling capacity of PM in the air, are most strongly affected by fine-mode elemental carbon particles, and do not directly measure mass or chemical composition of PM. Attempts to relate CoHs to μg/m^3 also require site-specific calibration of CoH readings against side-by-side gravimetric mass measurements, but the accuracy of such mass estimates is questionable and clearly only applicable for the particular location(s) where carried out for a limited time period.

The high volume (hi-vol) sampler method, used in the United States to measure TSP, directly measures the mass of the PM collected by gravimetric means. The D_{50} cutpoint for the sampler is typically around 25 to 50 μm, and collection of larger particles tends to drop off rapidly above such cutpoints. Thus, the hi-vol sampler, as typically employed, collects both fine- and coarse-mode particles that may include windblown crustal material of natural origin (especially in dry rural areas). Only rarely have cyclone samplers or other variations of the hi-vol sampler with smaller size cutpoints been used in epidemiological studies to limit collected particles to an inhalable range, but even then the cutpoints achieved were not sharp or independent of wind speed. Numerous factors other than wind speed, as discussed in Chapter 3, can also affect PM measurements by hi-vol sampling techniques. However, quality assurance information for TSP measurements reported in most American epidemiological studies is largely lacking, limiting statements that can be made about relative accuracy or precision of the TSP data reported.

The broader size range of particles sampled by the hi-vol versus the BS or ASTM methods severely limits intercomparisons of PM measurements by those methods to equivalent TSP units or vice versa. With few exceptions, no consistent relationship typically existed, for example, between BS and TSP measurements taken at various British sites or during various seasons at the same site (Commins and Waller, 1967; Lee et al., 1972; Ball and Hume, 1977;

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[*] For this reason, smoke data reported in μg/m^3 based on either the British or OECD Standard curve are generally most appropriately interpreted in terms of "nominal" μg/m^3 smoke units and cannot be accepted as accurate estimates of airborne PM mass unless corroborated by local site-specific gravimetric calibrations. In other words, unless based on local site-specific calibrations, smoke readings in μg/m^3 cannot yield quantitative estimates of atmospheric PM concentrations. Otherwise, such readings only allow for rough qualitative (i.e. <; =; or >) comparisons of amounts of PM present at a given time versus another time at the same site and generally do not permit meaningful comparisons between PM levels at different geographic areas having airborne PM of different chemical composition (especially in terms of relative porportions of elemental carbon).

Holland et al., 1979). One exception was during severe London air pollution episodes, when low wind speeds resulted in settling out of large coarse-mode particles and smaller particles increased to levels (>500 μg/m^3) such that BS and TSP measurements tended to converge (as expected when fine-mode and small coarse-mode particles predominate in the PM sampled).

Taking into account the foregoing information, aerometric data cited in published epidemiological studies must be viewed only as very approximate estimates of atmospheric levels of SO_2, particulate sulfur compounds, or other PM associated with reported health effects. Further, to the extent that (1) the cited aerometric data are derived from use of techniques with limited specificity for the substance(s) purportedly measured or (2) the relative contributions of PM or SO_2 to observed health effects cannot be distinguished from each other or from the effects of other covarying pollutants, then the aerometric data and associated health effects reported might be more appropriately viewed as relatively nonspecific indicators of the effects of air pollutant mixtures containing PM and SO_x.

1.14.3 Acute Exposure Effects

Detailed study of human health effects due to severe air pollution episodes spans a period of less than 50 years. The first reliable account of such episodes describes a 1930 incident in the Meuse Valley of Belgium. Dense fog covered the valley from December 1 to 5, with low winds and large amounts of PM present. About 6,000 residents became ill and 60 deaths associated with the fog occurred on December 4-5. The people who died were only briefly sick and the onset of acute illnesses abated rapidly when the fog dispersed. The death rate was 10.5 times normal. During a later event, when Donora, Pennsylvania, was blanketed by a dense fog in October 1948, 43 percent of the population of ~10,000 people was adversely affected. Twenty persons, mostly adults with preexisting cardiopulmonary diseases, died during or shortly after the fog due to cardiorespiratory causes. In a followup study, increased mortality rates and morbidity effects (e.g., heart disease and chronic bronchitis) were found among residents who reported acute illness during the 1948 episode in comparison to those reporting no acute illness. The Meuse Valley and Donora incidents demonstrated that severe air pollution can cause death and serious morbidity effects in human populations and raised the possibility of PM and SO_2 being among the causative agents.

As shown in Table 1-12, a series of episodes was also documented in London between 1948 and 1962. Excess mortality during those episodes occurred mainly among the elderly and chronically ill adults during periods of marked air pollution for several days. Various factors might help to explain the excess mortality, including possible influences not only of increased air pollution but also of high humidity (fog) and low temperatures. Regardless of the relative contributions of such factors, a clear consensus exists that increases in mortality were associated with air pollution episodes when 24-hr concentrations of both SO_2 and BS exceeded 1000 μg/m^3 in London; but the effects of specific pollutants acting alone or in combination cannot be clearly distinguished.

TABLE 1-12. EXCESS DEATHS AND POLLUTANT CONCENTRATIONS DURING SEVERE AIR POLLUTION EPISODES IN LONDON (1948-62)

Date	Duration, days	Deviation from $\bar{X}$ of total excess deaths*	Maximum 24-hr pollutant concentration, $\mu g/m^3$ Smoke (BS)	Maximum 24-hr pollutant concentration, $\mu g/m^3$ SO_2 (H_2O_2 titration)
Nov. 1948	6	750	2780	2150
Dec. 1952	4	4000	4460**	3830
Jan. 1956	4	1000	2830	1430
Dec. 1957	4	750	2417	3335
Jan. 1959	6	250	1723	1850
Dec. 1962	5	700	3144	3834

*Note that the numbers of excess deaths listed represent 15 to 350 percent increases in normal London baseline death rates during the years listed.

**Note that peak and 24-hr BS levels were likely much higher than 4460 $\mu g/m^3$ due to rapid saturation of filter paper by collected PM.

Source: Holland et al. 1979.

Acute episodes of high air pollution also occurred in the United States since the 1948 Donora episode, but no single event reached the magnitude of the London episodes. Some published studies (Greenburg et al., 1962, 1967; Glasser et al., 1967) suggested that increases in mortality may have occurred during certain New York City episodes in the 1950s and 1960s, when PM levels exceeded 5.0 to 8.0 CoHs and SO_2 exceeded 1000 $\mu g/m^3$, as measured at a single monitoring station in central Manhattan. Independent evaluation of the same New York City data led to one published report (McCarroll and Bradley, 1966) confirming apparent associations between increased mortality and acute episodes of high PM and SO_2. However, later reexamination of the New York data and the published analyses by the Greenburg group and by McCarroll and Bradley (1966) led Cassell et al. (1968) to question the validity of the earlier published conclusions, especially in view of difficulties in separating air pollution episode effects on mortality from effects of competing factors such as temperature and humidity extremes and epidemic illnesses, which appeared to exert much larger effects on death rates than the air pollution episodes. Still further doubts about the reported associations between New York City air pollution episodes and mortality are raised by inconsistencies in the data, such as no evident mortality increases being associated with some days of PM and/or SO_2 elevations as high or higher than those on other days reported to be associated with excess mortality. Thus, the results of the New York City episode studies do not provide much evidence for an association between increased mortality and episodic elevations of PM and SO_2.

When a marked increase in air pollution is associated with a sudden rise in the death rate or illness rate that lasts for a few days and when both return to normal shortly thereafter (as documented in some of the above studies), a causal relationship is strongly suggested. But sudden changes in weather, which may have caused the air pollution incidents, must also be considered as a possible cause of the death rate increase. However, the consistency of associations between SO_2 and PM elevations in London and increases in mortality make it very unlikely that weather changes alone provide an adequate explanation for all such observations. This view is further reinforced by: (1) some episodes not being accompanied by sharp falls in temperature; and (2) other weather changes of similar magnitude to those during the pollution episodes not being associated with dramatic mortality increases in the absence of increased levels of SO_2, PM, or other pollutants. In summary, the London episode studies provide clear evidence for substantial increases in excess mortality when the general population was exposed over several successive days to air pollution containing SO_2 concentrations $\geq 1000\ \mu g/m^3$ in the presence of PM levels over $1000\ \mu g/m^3$ (BS). Certain New York studies also tentatively suggest that small increases in excess mortality may have resulted from simultaneous elevations of SO_2 at $1000\ \mu g/m^3$ and PM at greater than 5.0-8.0 CoHs, but this is much less clearly established.

Comparison of the New York City episode data and those for the Meuse Valley, Donora, and London episodes reveals further important observations. Perhaps most striking are the much lower estimates of excess mortality reported for the New York episodes (at most 4 to 20 percent) compared to the 15 to 350 percent increases in death rate during the London episodes and even larger mortality rate increases in Donora and the Meuse Valley. Numerous factors might be cited to explain the striking differences, including likely variations in the specific chemical composition of the pollutant mixes present in the different areas and the much greater peak levels of pollutants (including PM and/or SO_2) that were probably present during the non-New York episodes. Also of probable considerable significance are two other features typifying the episodes in the Meuse Valley, Donora, and London: (1) the presence of extremely dense fog together with accumulating air pollutants, possibly providing the basis for transformation of pollutants to potentially more toxic forms (e.g., formation of sulfuric acid aerosol or absorption of PM into water droplet particles) resulting in more deposition of toxic substances in tracheobronchial regions of the respiratory tract and possible effects on mucociliarly clearance processes (see Chapters 11 and 13); and (2) the generally much more prolonged, continuous exposures of the non-New York populations to marked elevations of the pollutants. Examination of published New York City episode reports reveals that during such episodes the contributing temperature inversion conditions typically intensified during evening hours, thus accumulating air pollutants overnight. However, the inversions dissipated during morning hours, thereby resulting in much higher peaks in PM and SO_2 in the mornings than in the afternoons (when PM and SO_2 levels fell back to near-normal levels). This is in contrast to the continuously high pollutant and fog levels that apparently persisted for several (4 or more) successive days during the Meuse Valley, Donora, and London episodes, with largest increases in mortality tending to occur on later days of each episode. Thus, although 24-hr concentrations of PM and SO_2 $\geq 1000\ \mu g/m^3$ can be stated as levels at which mortality has notably increased, great care must be exercised in generalizing from these observations in attempting to predict likely effects associated with comparable concentrations at other times and locations. In particular, the prolonged or continuous nature of the high pollutant exposures and other interacting factors, e.g., high humidity levels, must be taken into account as additional important determinants of mortality increases observed so far during major air pollution episodes. Moreover, marked increases in mortality should not be expected to occur regularly as a function of short-term peak excursions of 24-hr PM or SO_2 levels barely exceeding 1000 $\mu g/m^3$. Consistent with this statement are numerous examples in the epidemiological literature where no detectable increases in mortality were found to occur on scattered days when PM and/or SO_2 levels reached comparably high ($\geq$1000 $\mu g/m^3$) 24-hr levels as on other days (or sets of successive days) when mortality was more clearly increased.

Even more difficult to establish are to what extent smaller but significant increases in mortality and morbidity are associated with nonepisodic 24-hr average exposures to SO_2 and/or PM levels below 1000 $\mu g/m^3$. Concisely summarized in Table 1-13 are findings from several key studies reviewed in Chapter 14 which appear to demonstrate with a reasonably high degree of certainty mortality and morbidity effects associated with acute (24-hr) exposures to these pollutants. The first two studies cited, by Martin and Bradley (1960) and Martin (1964), deal with a relatively small body of data from London in the late 1950s. No clear "threshold" levels were revealed by their analyses regarding SO_2 or BS levels at which significantly increased mortality began to occur. However, based on their findings and a reanalysis of the Martin and Bradley data by Ware et al. (1981), mortality in the elderly and chronically ill was clearly elevated in association with exposure to ambient air containing simultaneous SO_2 and BS levels above 1000 $\mu g/m^3$; and some indications exist from these analyses that slight increases in mortality may have been associated with nonepisodic BS and PM levels in the range of 500 to 1000 $\mu g/m^3$ (with greatest certainty demonstrated for levels in excess of 750 $\mu g/m^3$). Much less certainty is attached to suggestions of mortality increases at lower levels possibly based on the Ware et al. (1981) or other reanalyses (Appendices 14D and 14E, Chapter 14) of the Martin and Bradley data, especially in view of wide 95 percent confidence intervals demonstrated by the reanalyses to be associated with estimation of dose-response relationships between mortality and BS or SO_2 using the Martin and Bradley (1960) data. Analyses by Mazumdar et al. (1981) for 1958-59 to 1971-72 (Figure 1-20) are generally consistent with the above findings but seem to suggest that the 1958-59 London winter may represent a worst-case situation in comparison to most later winters. Still, the Mazumdar et al. (1981) and certain other analyses (Appendix 14E, Chapter 14) of 1958-59 to 1971-72 London winter mortality data are strongly indicative of small, but significant, increases in mortality occurring at BS levels below 500 $\mu g/m^3$ and, possibly, as low as 150 to 200 $\mu g/m^3$.

Only very limited data exist by which to attempt to delineate any specific physical and chemical properties of PM associated with the observed increases in mortality. Taking into consideration information noted earlier (Section 1.14.2), marked increases in fine-mode and small coarse-mode particles to levels above 500-1000 $\mu g/m^3$ appear, based on the reported BS aerometric measurements, to be most clearly associated with increased mortality, although

TABLE 1-13. SUMMARY OF QUANTITATIVE CONCLUSIONS FROM EPIDEMIOLOGICAL STUDIES RELATING HEALTH EFFECTS TO ACUTE EXPOSURE TO AMBIENT AIR LEVELS OF SO_2 AND PM

Type of study	Effects observed	24-hr average pollutant level ($\mu g/m^3$) BS	SO_2	Reference
Mortality	Clear increases in daily total mortality or excess mortality above a 15-day moving average among the elderly and persons with preexisting respiratory or cardiac disease during the London winter of 1958-59.	$\geq$1000	$\geq$1000	Martin and Bradley (1960); Martin (1964)
	Analogous increases in daily mortality in London during 1958-59 to 1971-72 winters.			Mazumdar et al. (1981)
	Some indications of likely increases in daily total mortality during the 1958-59 London winter, with greatest certainty (95% confidence) of increases occurring at BS and SO_2 levels above 750 $\mu g/m^3$	500-1000	500-1000	Martin and Bradley (1960)
	Analogous indications of increased mortality during 1958-59 to 1971-72 London winters, again with greatest certainty at BS and SO_2 levels above 750 $\mu g/m^3$ but indications of small increases at BS levels <500 $\mu g/m^3$ and possibly as low as 150-200 $\mu g/m^3$.			Mazumdar et al. (1981)
Morbidity	Worsening of health status among a group of chronic bronchitis patients in London during winters from 1955 to 1960.	$\geq$250-500*	$\geq$500-600	Lawther (1958); Lawther et al. (1970)
	No detectable effects in most bronchitics; but positive associations between worsening of health status among a selected group of highly sensitive chronic bronchitis patients and London BS and SO_2 levels during 1967-68 winter.	<250*	<500	Lawther et al. (1970)

*Note that the 250-500 $\mu g/m^3$ BS levels stated here may represent somewhat higher PM concentrations than those actually associated with the observed effects reported by Lawther et al. (1970). This is because their estimates of PM mass (in $\mu g/m^3$ BS) were based on the D.S.I.R. calibration curve found by Waller (1964) to approximate closely a site-specific calibration curve developed by Waller in central London in 1956, but yielding somewhat higher mass estimates than another site-specific calibration developed by Waller a short distance away in 1963. However, the precise relationship between estimated BS mass value based on the D.S.I.R. curve versus the 1963 Waller curve cannot be clearly determined due to several factors, including the non-linearity of the two curves and their convergence at low BS reflectance levels.

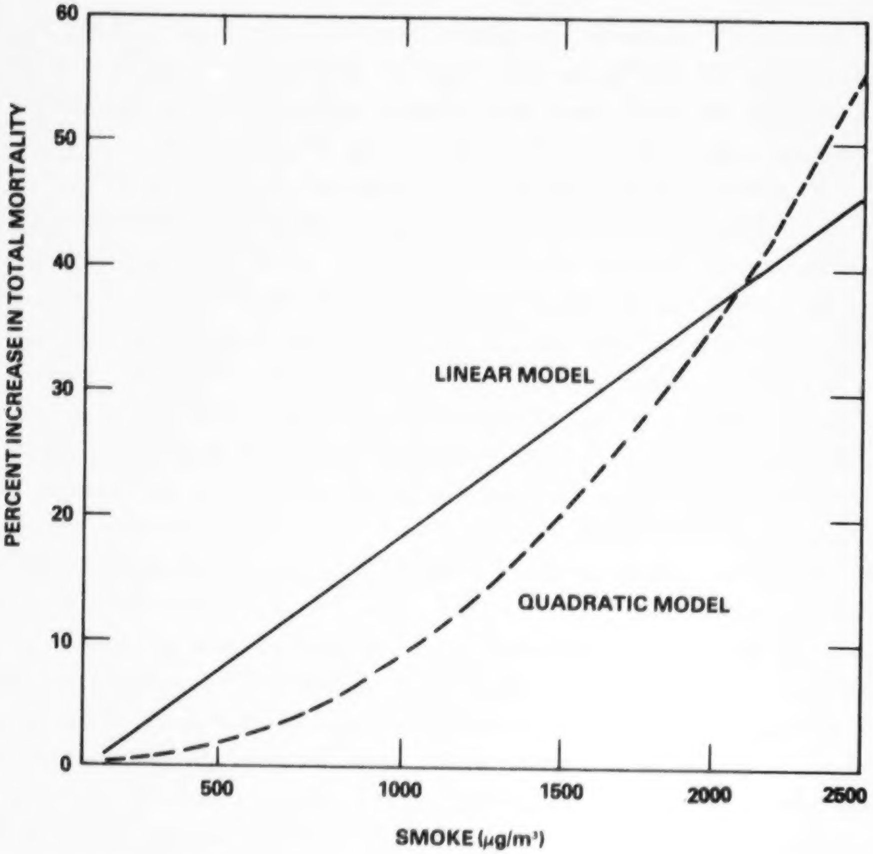

Figure 1-20. **Hypothetical dose-response curves derived from regressing mortality on smoke in London, England during winters 1958/59 to 1971/72. Results obtained with linear (—) and quadratic (- -) models are depicted for comparison.**
Source: Mazumdar et al. (1981).

contributions from larger coarse-mode particles cannot be completely ruled out. Nor is it possible to state with certainty which PM chemical species were associated with the increases in mortality. It is known that large amounts of pollutants (e.g., elemental carbon, tarry organic matter, etc.) from incomplete combustion of coal were present in the air, and mortality levels appeared to decrease as PM concentrations declined over the years; but no single component or combinations of particulate pollutants can clearly be implicated. Neither can the relative contributions of SO_2 or PM be clearly separated based on these study results, nor can possible interactive effects with increases in humidity (fog) be completely ruled out. Temperature change, however, does not appear to be a key determinant in explaining mortality effects demonstrated by the above analyses to be associated with atmospheric elevations of PM or SO_2.

Analysis of the Lawther morbidity studies listed in Table 1-13 suggests that acute exposure to elevated 24-hr PM levels in the range of 250-500 $\mu g/m^3$ (BS) in association with 24-hr SO_2 levels of 500-600 $\mu g/m^3$ were most clearly associated with exacerbation of respiratory disease symptoms among large (>1000) populations of chronically ill London bronchitis patients. Most such patients were apparently not affected at lower BS or SO_2 levels. However, a smaller population (~80) of selected, highly sensitive London bronchitis patients appeared to be affected at somewhat lower BS and SO_2 levels, but specific exposure-effect levels cannot be determined on the basis of the reported data. Again, little can be said, however, in terms of specifying physical or chemical properties of PM associated with the observed effects beyond the comments noted above in relation to Martin's mortality studies.

Other studies, besides those of Lawther, tend to suggest that the elderly, people with chronic cardiorespiratory diseases, and children may constitute populations at risk for manifesting morbidity effects in response to acute exposure to elevated atmospheric levels of SO_2 and PM. Qualitatively, increases in the occurrence of cardiac and upper respiratory tract disease symptoms, including exacerbation of preexisting chronic bronchitis (but not asthma attacks), appear to be among the morbidity effects most clearly associated with exposures to the ambient levels of PM and SO_2 evaluated in those studies and are most clearly seen at markedly elevated levels of the two pollutants. For example, increased applications by adults aged 45-79 for admissions to London hospitals for cardiac and respiratory morbidity most clearly occurred, based on the Martin (1964) study, when 24-hr BS and SO_2 levels approached or exceeded 900-1000 $\mu g/m^3$; but Martin's data also suggest that such effects may have occurred at somewhat lower levels, i.e., down to 500 $\mu g/m^3$ for both SO_2 and BS. Similarly, American studies by Greenburg's group appear to most clearly suggest increased cardiac and upper respiratory morbidity, especially among the elderly, during air pollution episodes in New York City when extremely high levels of PM (5.0-8.0 CoHs) and SO_2 (>1000 $\mu g/m^3$) were present. On the other hand, much less clearly demonstrated were morbidity effects related to nonepisodic elevations in New York City of air pollution containing PM and SO_2. The findings of McCarroll's group (especially as reported by Lebowitz et al., 1972), for example, suggest at most an increase in upper respiratory tract symptoms (e.g., coughs and colds) in certain "sensitive" children at lower nonepisodic levels of PM or SO_2 in New York City. Insufficient epidemiological information from such studies exists, however, by which to determine specific quantitative acute exposure levels at which such "sensitive" children may have been affected.

1.14.4 Chronic Exposure Effects

Numerous studies have been performed to compare general or cause-specific mortality in areas of lowest-to-highest pollution concentrations. However, virtually all of these studies (1) used aerometric data of questionable accuracy or representativeness of study population exposures, and (2) did not adequately account for the potential effects on mortality rates of such confounding factors as cigarette smoking, occupation, social status, or mobility differences between areas (see Appendix 14A, Chapter 14). These methodological problems preclude accurate characterization of any quantitative relationships between mortality and air pollution parameters. Therefore, essentially no epidemiological studies are presently well-accepted as providing valid quantitative data relating respiratory disease or other types of mortality to chronic (annual average) exposures to PM or SO_x. On the other hand, the findings of certain published studies of chronic air pollution effects on mortality appeared to warrant consideration in regard to their potential for establishing qualitative links between mortality and chronic exposures to PM or SO_x. Two types of general approaches were employed in such studies: (1) aggregation of mortality and other information, e.g. smoking or socioeconomic status data, in relation to specific individuals within the study population(s); and (2) aggregation of analogous data for entire populations across large geographic areas, e.g. cities, counties, or standard metropolitan statistical areas.

Among the best known examples of the first approach are the Winkelstein et al. (1967), Winkelstein and Kantor (1967), and Winkelstein and Gay (1971) studies of total and cause-specific mortality in Buffalo and Erie County, New York, during 1959 to 1961. A network of 21 sampling stations provided data on TSP (hi-vol sampler) and oxides of sulfur (non-specific sulfation methods) for the period July 1961 to June 1963; and these aerometric data were used to categorize geographic areas as "low" to "high" air pollution areas. Chronic respiratory disease mortality for white males 50 to 69 years old was reported to be about three times higher in the high-pollution areas than in the low-pollution areas, across all economic groups (Winkelstein et al., 1967). Additional positive associations in relation to TSP concentrations were reported for both stomach cancer (Winkelstein and Kantor, 1967) and deaths from cirrhosis of the liver (Winkelstin and Gay, 1971). However, numerous criticisms can be noted which raise serious doubts regarding the validity of the reported findings, including the following methodological problems: (1) the use of 1961-1963 TSP and SO_x measurement data as a basis for retrospectively classifying geographic areas according to presumed past air pollution gradients against which to compare mortality among the elderly during 1959 to 1961; (2) inadequate controls for possible age differences between study groups that may have covaried with the air pollution gradient used; (3) lack of information on lifetime (including occupational) exposures to PM or SO_2; (4) failure to correct for smoking habits; and (5) the implausibility of some of the reported findings, e.g., air pollution increasing mortality due to liver cirrhosis. Later, Winkelstein (1972) attempted to correct for some of these problems by looking at smoking patterns among populations living in the same study areas included in the earlier studies, but the 1972 analyses do not adequately counter major concerns about the earlier studies. For example, the reported 1972 follow-up investigation found no significant differences in smoking patterns among the different study areas for females, but this finding does not adequately control for possible smoking effects in different specific population cohorts evaluated in the earlier studies. These studies, therefore, are of questionable validity in regard to providing credible qualitative evidence for links between PM air pollution and mortality.

The second type of approach listed as being used for evaluation of chronic air pollution effects on mortality is typified by the work of Lave and associates. Lave and Seskin (1970) carried out regression analyses on relationships between PM air pollution (indexed by deposit gauges and BS measurements) in Britain and bronchitis mortality data, taking into account the effects of socioeconomic status (SES). They reported positive associations between such mortality and PM pollution. However, the Lave and Seskin (1970) study has been extensively criticized in detail by others who noted difficulties in justifying inclusion of SES and air pollution levels in the analyses as if they were completely independent variables and failure to make direct allowance for smoking habits in the analyses. Still more basic difficulties with the analyses derive from: (1) use of qualitative BS aerometric data expressed in terms of mass concentration estimates (in $\mu g/m^3$) not appropriately obtained by means of site-specific calibrations of reflectance readings against local gravimetric mass data; and (2) ambiguities regarding locations of sampling devices in relation to study population residences, which raise serious questions regarding the representativeness of the aerometric data used in estimating population PM exposures.

In three later publications (Lave and Seskin, 1972, 1977; Chappie and Lave, 1981), the results of further extension of their cross-sectional analysis approach (Lave and Seskin, 1970) to standard metropolitan statistical areas (SMSAs) in the United States were reported. Significant positive associations between mortality and certain air pollution variables (e.g., TSP and sulfate levels) were reported for 1960, 1969, and/or 1974 U.S. data, suggesting that air pollution variables made a significant contribution to explaining differences in mortality rates among the SMSAs. However, based on their analyses, it was not possible to quantify the individual contributions of each air pollutant and other variables to the observed mortality rates. Many criticisms similar to those indicated above for the earlier Lave and Seskin (1970) publication apply here. Of crucial importance are basic difficulties associated with all of their analyses in terms of: (1) use of aerometric data without regard to quality assurance considerations, including use of sulfate measurements known to be of questionable accuracy due to artifact formation during air sampling (see Sections 1.3 and 1.14.2); (2) questions regarding the representativeness of the air pollution data used in the analyses as estimates of actual exposures of individuals included in their study populations;

and (3) overgeneralization of findings in extrapolating results obtained for limited air pollutant levels or selected localities across much broader ranges of pollutants and geographic areas despite indications to the contrary. Clearly, then, no useful information on quantitative relationships between specific concentrations of PM or SO_x and mortality can be derived from these published analyses. Similarly, only very limited qualitative conclusions can be stated regarding PM or SO_x air pollution-mortality relationships, based on the results of these and other analogous "macroepidemiological" studies, as discussed in Chapter 14.

In regard to morbidity effects associated with chronic exposure to PM and SO_2, the best pertinent epidemiological health studies are summarized in Table 1-14. The studies by Ferris et al. (1973, 1976) suggest that lung function decrements may occur in adults at TSP levels in excess of 180 $\mu g/m^3$ in the presence of relatively low estimated SO_2 levels, whereas no effects were observed by the same investigators at TSP levels below 130 $\mu g/m^3$. Other studies (Lunn et al., 1967) listed in Table 1-14 suggest that significant respiratory effects occur in children in association with long-term (annual average) PM levels in the approximate range of 230-301 $\mu g/m^3$ (BS) in association with SO_2 levels of 181-275 $\mu g/m^3$, although no clearly distinct thresholds are evident (see Figure 1-21). A later 3-year followup study (Lunn et al., 1970) of cohorts of children from the same study population (in Sheffield, England), however, failed to find demonstrable respiratory effects attributable to air pollution following marked decreases in PM and SO_2 levels. This suggests possible recovery from earlier-detected respiratory disease symptoms and associated decrements in pulmonary function as a result of decreased exposure to PM or SO_2.

No particular PM chemical species can clearly be implicated as causal agents associated with the effects observed in the studies listed in Table 1-14. Nor can potential contributions of relatively large inhalable coarse-mode particles be ruled out on the basis of these study results. It should be remembered that various occupational studies listed in Appendix 14B of Chapter 14 at least qualitatively suggest that such sized particles of many different types of chemical composition can be associated with significant pulmonary decrements, respiratory tract pathology, and morphological damage--at least at relatively high exposure levels.

Only very limited information has (summarized in Table 1-15) been published (Commins and Waller, 1967) on the chemical composition of particulate matter present in London air during the period of some of the above epidemiological studies of associations between mortality or morbidity effects and elevations in PM levels. Such data may provide important clues as to possible causative agents involved in the etiology of health effects observed in London during the 1950s and early 1960s. For the sake of comparison, information on measured chemical components of TSP matter in U.S. cities during the early 1960s is also provided in Table 1-15. It must be noted, however, that likely substantial differences in specific components of the PM present in London air of the 1950s and 1960s versus the chemical composition of PM currently present in urban aerosols over American cities argue for much caution in

TABLE 1-14. SUMMARY OF QUANTITATIVE CONCLUSIONS FROM EPIDEMIOLOGICAL STUDIES RELATING HEALTH EFFECTS TO CHRONIC EXPOSURE TO AMBIENT AIR PM AND SO_2

Type of study	Effects observed	Annual average pollutant levels ($\mu g/m^3$) particulate matter BS	TSP	SO_2	Reference
Cross-sectional (4 areas)	Likely increased frequency of lower respiratory symptoms and decreased lung function in children in Sheffield, England	230-301*	-	181-275	Lunn et al. (1967)
Longitudinal and cross-sectional	Apparent improvement in lung function of adults in association with decreased PM pollution in Berlin, NH	-	180	**	Ferris et al. (1973, 1976)
Longitudinal and cross-sectional	Apparent lack of effects and symptoms, and no apparent decrease in lung function in adults in Berlin, NH	-	80-131	**	Ferris et al. (1973, 1976)

*Note that BS levels stated here in $\mu g/m^3$ must be viewed as only crude estimates of the approximate PM (BS) mass levels associated with the observed health effects, given ambiguities regarding the use or non-use of site-specific calibrations in Sheffield to derive the reported BS levels in $\mu g/m^3$.

**Note that sulfation rate methods indicated low atmospheric sulfur levels in Berlin, NH during the time of these studies. Crude estimation of SO_2 levels from that data suggest that SO_2 levels were generally <25-50 $\mu g/m^3$ and did not likely contribute to observed health effects.

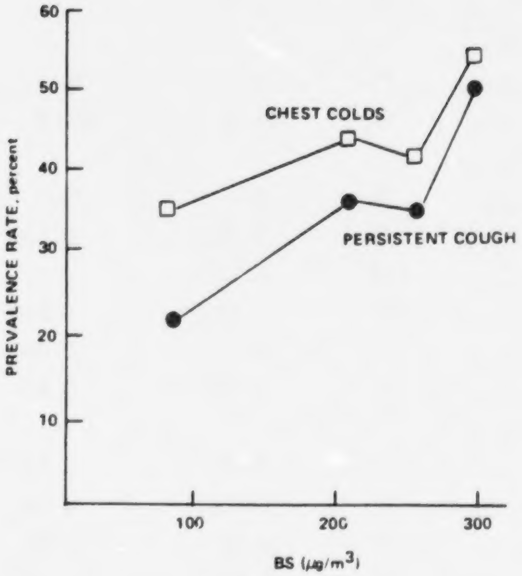

Figure 1-21. History and clinical evidence of respiratory disease (percent) in 5-year-olds, by pollution in area of residence. BS ($\mu g/m^3$) levels indicated above must be taken as only very crude approximations of actual PM mass present due to ambiguities regarding use of site-specific calibrations in deriving the mass estimates.

Source: Lunn et al. (1967).

TABLE 1-15. COMPARISON OF MEASURED COMPONENTS OF TSP IN U.S. CITIES (1960-1965) AND MAXIMUM 1-HOUR VALUES IN LONDON (1955-1963)

	UNITED STATES[a]			LONDON[b]
		Concentration μg/m^3		
Pollutant	Number of stations	Arith. average	Maximum 24-hour	Maximum 1-hour[c]
Suspended Particles	291	105	1254 (TSP)	9700 (Smoke)
Fractions:				
Benzene-soluble organics	218	6.8	-	-
Chloride (water soluble)	-	-	-	410
Nitrates	96	2.6	39.7	5
Sulfates	96	10.6	101.2	666
Sulfuric acid	-	-	-	680
Ammonium	56	1.3	75.5	-
Antimony	35	0.001	0.160	<1
Arsenic	133	0.02	-	-
Beryllium	100	<0.0005	0.010	<1
Bismuth	35	<0.0005	0.064	<1
Cadmium	35	0.002	0.420	1
Calcium	-	-	-	32
Chromium	103	0.015	0.330	2
Cobalt	35	<0.0005	0.060	<1
Copper	103	0.09	10.00	2
Iron	104	1.58	22.00	25
Lead	104	0.79	8.60	22
Manganese	103	0.10	9.98	5
Molybdenum	35	<0.005	0.78	<1
Nickel	103	0.034	0.460	1
Tin	85	0.02	0.50	2
Titanium	104	0.04	1.10	1
Vanadium	99	0.050	2.200	2
Zinc	99	0.67	58.00	24
Gross beta radioactivity	323	(0.8 pCi/m^3)	(12.4 pCi/m^3)	

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[a] U.S. Department of Health, Education, Welfare (1970)

[b] Commins and Waller (1967)

[c] Obtained from one London site.

extrapolating results of London epidemiological studies for present criteria development purposes.

1.14.5 Implications Of Epidemiological Findings For Criteria Development Purposes

Several epidemiological investigations of health effects associated with exposures to PM and SO_2 in London during the 1950s and 1960s (summarized in Table 1-13) appear to provide a reliable basis by which to estimate quantitatively ambient air levels of PM and SO_2 at which acute exposure effects are likely to be seen, under some circumstances, among certain human population groups at special risk. More specifically, the elderly and those with preexisting cardiorespiratory disease conditions appear to be at greatest risk for acute PM and SO_2 exposure effects, based on the London mortality and morbidity studies summarized in Table 1-13. As noted above, however, great care must be exercised in extrapolating from the observed exposure-effect relationships indicated in Table 1-13 to what might be expected to occur at other times or geographic locations. That is, acute exposure effects of the type listed in the table may not occur at the indicated pollutant levels under different meteorological conditions or with varying atmospheric aerosols that differ substantially in particle size and chemical composition from those present in London during the 1950s and 1960s. High humidity levels (fog conditions) occurring jointly with prolonged simultaneous elevations of PM and SO_2, for example, may be required before the most marked mortality effects listed in Table 1-13 would occur.

In relation to aerosol composition, as noted earlier, it is not possible to delineate precisely specific particle sizes or chemical species that may have been crucial in inducing the observed health effects noted in Table 1-13. Only reasonable possibilities can be deduced from the available epidemiological data and other types of information presented elsewhere in the present document. For example, concerning the size of particles likely associated with observed health effects, both mortality and morbidity effects increased in relation to elevations in PM concentrations as indexed by BS measurements. Recently, McFarland et al. (1982) demonstrated that the BS sampling apparatus, as typically employed in the field, was capable of collecting particles up to about 7-9 μm MMAD, with 50 percent efficiency for ~ 4.5 μm-sized particles, under low (2 km/hr) wind-speed conditions (see Figure 1-22). Variations in exact configurations of BS sampler apparatus inlet tubing in the field and other conditions (e.g., different wind speeds) present at the time of actual BS sampling in London during the 1950s and 1960s, however, likely resulted in some deviations (both higher and lower) in collection efficiencies for various size particles in comparison to those depicted in Figure 1-22. Nevertheless, it appears that, in general, particles less than 7-9 μm were sampled by the BS apparatus, with greatest efficiency for those below 4-5 μm MMAD.

In light of the above, the mortality and morbidity effects found by studies summarized in Table 1-13 to be associated with increases in BS levels might be most reasonably and directly attributed to fine- and small coarse-mode particles of <7-9 μm MMAD. This would be consistent with the potential for respiratory effects occurring as the result of significant

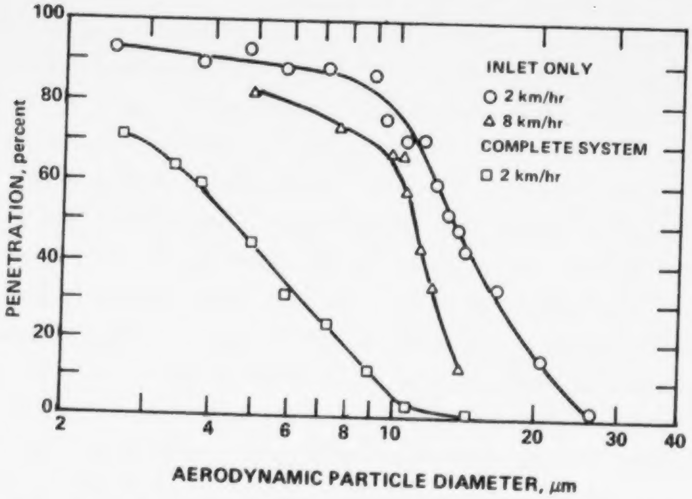

Figure 1-22. Penetration of aerosol through the inlet of the British Smoke Shade Sampler and through the complete system.

Source: McFarland et al. (1982).

proportions of fine- and coarse-mode particles (<10-15 μm MMAD) being deposited in thoracic (i.e., tracheobronchial and pulmonary) regions of the respiratory tract with mouth breathing, as demonstrated by deposition studies summarized in Figure 1-19. It is unfortunate, however, that more precise estimates of concentrations of particles in the thoracic particle (TP) range (i.e., <10-15 μm MMAD) present in London air during the periods studied by the epidemiological investigations listed in Table 1-13 do not exist, as would have been measured better by presently available modified hi-vol sampling devices with relatively sharp 10- or 15-μm cutpoints (see Section 1.3 and Chapter 3). As it is, no simple, precise, or invariable relationship(s) can be stated between atmospheric TP concentrations and PM levels indexed by BS measurements demonstrated by epidemiological studies listed in Table 1-13 to be associated with mortality and morbidity effects in London of the 1950s and 1960s. Nor can there now be stated any precise, consistent relationships between such TP levels and fine-particle (<2.5 μm) mass or TSP (<25-50 μm) mass, as measured by presently available dichotomous or hi-vol samplers of types alluded to in Section 1.3 or Chapter 3. However, based on present knowledge, it would appear that the following relationships are, in general, probably correct: fine-particle mass < reported London BS values expressed in μg/m^3 < TP mass (as defined by particles < 10 - 15 μm MMAD) < TSP mass. Further, based on recently reported observations by Pace et al. (1981), comparing seasonal variations in concentrations of fine-mode (<2.5 μm) particle, inhalable (<15 μm) particle, and TSP (<25-50 μm) particle mass in several Eastern and Midwestern U.S. cities, it appears that TP mass may generally constitute roughly 40 to 60 percent of TSP mass currently found in atmospheric aerosols over many U.S. cities.

In regard to chronic PM and SO_2 exposure-effect relationships indicated by studies summarized in Table 1-14, it should be noted that the Lunn et al. (1967) study demonstrates that increased risk for respiratory symptoms and pulmonary function decrements among young school age children are associated with long-term chronic exposures to the PM and SO_2 levels listed in the table. However, no clear threshold levels can clearly be discerned based on the Lunn et al. (1967) study results, such that some small but undefined degree of risk might exist at or below the lowest pollutant levels depicted for the "control" study population in Figure 1-21. On the other hand, the possibility of any increased risk existing at such exposure levels can neither be scientifically confirmed nor denied in the absence of additional data. The lack of any detectable similar effects being found 3 years later (in a followup study by the same investigators) among other children of the same age or cohorts of the same children studied earlier tends to suggest, however, that such risks are likely nonexistent or minimal at annual average PM or SO_2 levels lower than those listed in Table 1-14. The same comments presented above regarding possible relationships between BS values listed in Table 1-13 and TP mass levels possibly associated with listed health effects also apply here for Table 1-14.

In regard to the other chronic exposure studies (by Ferris et al.) listed in Table 1-14, it should be noted that the results reported are for relatively small study cohorts investigated for brief intervals of time over the course of several years. Also, the improvements in lung functions (as measured by spirometric methods) in study subjects from one time point to another (coincident with decreases in TSP levels during the same time periods as indicated by limited air monitoring data) represent only a rather modest basis upon which to attempt to estimate ambient air PM levels at which health effects are likely to occur in the general population.

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Newly Available Information on Health Effects Associated with Exposure to Sulfur Dioxide as Evaluated by Controlled Human Exposure Studies: An Addendum to the EPA Criteria Document Entitled *Air Quality Criteria for Particulate Matter and Sulfur Oxides* (December, 1982)

The EPA document entitled "Air Quality Criteria for Particulate Matter and Sulfur Oxides" (to which this addendum is appended) was substantively completed in December, 1981, and made available for use in decision making regarding possible revision by EPA of National Ambient Air Quality Standards (NAAQS) for particulate matter (PM) and sulfur dioxide (SO_2). Since the 1981 completion of the Criteria Document and during its editorial preparation for publication in its present form (December, 1982), several scientific articles, newly published or accepted for publication in peer-reviewed journals, have become available and appear to provide important information pertinent to development of criteria for primary (health-related) NAAQS for SO_2. This addendum to the Criteria Document summarizes and evaluates the newly available studies and attempts to place their findings in perspective in relation to the results of certain other key studies and conclusions discussed in Chapter 13 of the Criteria Document. This includes discussion of data and conclusions bearing on such important issues as:

(1) SO_2 exposure levels associated with the induction of pulmonary mechanical function effects (e.g., bronchoconstriction) in sensitive individuals under increased activity conditions;

(2) Mechanisms of action by which such pulmonary function effects may be mediated in sensitive individuals;

(3) Possible enhancement in sensitive individuals of SO_2-induced pulmonary function effects by combined SO_2-PM aerosol exposures.

In relation to the first issue, various studies discussed in Chapter 13 of the Criteria Document indicate that the level of physical activity of human subjects (both nonsensitive and sensitive individuals) is an important determinant of SO_2 exposure concentrations at which measurable changes in pulmonary function and symptomatic effects are manifested. This is mainly due to the fact that most human subjects, while at rest, breathe nasally (i.e., through the nose), where more than 90 percent of inhaled SO_2 is normally absorbed by the nasal mucosa and does not penetrate deeper into tracheobronchial regions of the respiratory tract. In contrast, with increased levels of physical activity or exercise, human subjects eventually reach a point where they shift over to oronasal breathing, during which time up to 40-50 percent of the inhaled air enters via the mouth and allows for substantial amounts of SO_2 to bypass nasal defense mechanisms and reach tracheobronchial regions of the respiratory tract (Niinimaa et al., 1981). The exercise level at which such a shift to oronasal breathing occurs varies widely for different subjects, the mean minute volume (V_e) at which such a shift occurs being 35.3 ± 10.8 ℓ/min. Such ventilation rates are equivalent to those experienced while engaged in moderately strenuous day-to-day physical activities, e.g. walking at a fast pace for some individuals or jogging for others, climbing a flight or two of stairs, or lifting and carrying relatively lightweight packages or other materials.

As discussed in Chapter 13 of the Criteria Document, various published studies indicate that most nonsensitive, healthy adult subjects do not experience pulmonary function changes or symptomatic effects (e.g. dyspnea, chest pains, etc.) at SO_2 levels below 5 ppm. However, with increased delivery of SO_2 to tracheobronchial regions of the respiratory tract, due either to forced oral breathing via a mouthpiece or increased oronasal breathing under light to heavy exercise conditions, nonsensitive adult subjects have been reported to experience pulmonary mechanical function changes at SO_2 exposure levels of 3 ppm or, in some cases, at levels as low as 0.75 ppm under heavy exercise conditions without a mouthpiece. In other studies (Sheppard et al.,1981b) certain sensitive population group subjects, i.e. individuals with clinically defined mild asthma, were shown to be about an order of magnitude more sensitive than the nonsensitive individuals. That is, statistically significant increases in airway resistance (SR_{aw}) indicative of bronchoconstriction and associated symptomatic effects were reported to occur in such subjects (as a group) at 0.5 ppm SO_2 under conditions of light exercise ($V_e \sim$ 30 ℓ/min) and forced oral breathing via a mouthpiece; and in some of the most sensitive individuals, SR_{aw} increases were reported at SO_2 levels as low as 0.1 ppm. Qualitatively similar results were independently obtained in a pilot-study by other investigators (Linn et al., 1982a) with forced oral breathing of 0.5 ppm SO_2 by mild asthmatics under light exercise conditions (at $V_e \sim$ 27 ℓ/min).

The possibility was raised, then, that bronchoconstriction might be experienced by mild asthmatic subjects in response to ambient air SO_2 exposures at levels below 1.0 ppm. However, direct extrapolation of specific dose-effect levels established in these controlled human exposure experiments to ambient situations was not possible due to the use of an artifical airway (mouthpiece) in these studies whereby the efficient SO_2 removal processes in the nasal passages are bypassed. Moreover, airflow characteristics of the oral airway show very marked differences between breathing through a mouthpiece and breathing through the spontaneous positioned mouth (Cole et al., 1982), lending support to the suggestion that SO_2 removal in the oral cavity during mouthpiece breathing is less efficient than that which occurs for unencumbered oronasal breathing. Thus, additional research would be necessary, using non-mouthpiece SO_2 exposure methods more closely approximating natural oronasal breathing, before dose-effect relationships established by controlled human exposure experiments could be extrapolated to ambient conditions.

In an initial study evaluating the effects of non-mouthpiece exposure to SO_2 on exercising asthmatic subjects, Linn et al. (1982a) exposed 24 adult mild asthmatic subjects (11 females and 13 males ranging in age from 21 to 27 years) to 0, 0.25, and 0.5 ppm SO_2 while engaged in unencumbered breathing during exercise in an open chamber. The SO_2 exposures, conducted in a controlled-exposure chamber at 23°C and R.H. = 90% or higher, lasted one hour during which 10-min. periods of exercise (mean $V_e \cong 27$ ℓ/min) were alternated with 10-min. rest periods. No statistically significant increases in SR_{aw} or associated symptoms were found in the mild asthmatic subjects with the open chamber exposures either to 0.25 or 0.5 ppm SO_2 under the light exercise conditions employed by Linn et al. (1982a). However, given a mean V_e of 27 ℓ/min., it is highly probable that the exercise conditions used were not sufficiently high to assure a shift to oronasal breathing by the study subjects and most of them probably breathed predominately nasally during SO_2 exposure while exercising. This study, then, left unresolved the issue of whether or not significant bronchoconstriction or symptomatic effects could be induced by SO_2 in mild asthmatic subjects under unencumbered oronasal breathing conditions simulating ambient circumstances.

In another study providing important evidence bearing on this issue, Kirkpatrick et al. (1982) compared pulmonary function and symptomatic effects obtained with SO_2 exposure of exercising mild asthmatic subjects via: (a) oral breathing; (b) oronasal breathing; or (c) nasal breathing. More specifically, Kirkpatrick et al. (1982) studied six non-smoking young adult subjects (4 men, 2 women), with medical histories suggestive of asthmatic disease but neither receiving medication nor recently exhibiting respiratory disease symptoms. These individuals exercised on a bicycle ergometer for 5 min. at 550 kpm/min which resulted in minute ventilation rates that averaged 41-44 ℓ/min ± 5.0-6.9 S.D. during different exposure conditions that included exposure to: (a) humidified air via mouthpiece; (b) humidified air plus 0.5 ppm SO_2 via mouthpiece with nasal airways obstructed (oral breathing); (c) humidified air plus 0.5 ppm SO_2 via a facemask (oronasal breathing); and (d) humidified air plus 0.5 ppm SO_2 via facemask but with mouth occluded (nasal breathing). Dose-response curves were additionally defined for two subjects exposed to 0, 0.25, 0.50 and 1.0 ppm SO_2 or to 0, 0.50, 1.0 and 2.0 ppm SO_2 via mouthpiece (orally) and facemask (oronasally). Both V_{tg} and SR_{aw} were measured before and after exercise for all six subjects; and the four highest consecutive baseline SR_{aw} values for each subject were compared with their four highest consecutive post-exposure SR_{aw} values for each exposure condition, using unpaired t-tests. In addition, further statistical evaluations of the group response under the different exposure conditions were made by analyses of variance (ANOVA's). The increase in SR_{aw} resulting from breathing SO_2 orally was significantly ($P < 0.01$) greater than the increase observed after breathing humidified air orally. SR_{aw} was also significantly greater when breathing SO_2 either by the oronasal or nasal routes. An independent analysis of variance confirmed that SO_2 inhalation by these asthmatic subjects produced bronchoconstriction regardless of the mode of entry into the lungs (personal communication from Horvath, 1982). For the group, although the increase in SR_{aw} was greater when subjects breathed SO_2 through a mouthpiece (oral) than when they breathed SO_2 from a facemask (oronasal), the difference did not achieve statistical significance at $P < 0.05$. Specific symptomatic responses (e.g. eye, nose and throat irritation or shortness of breath and cough), were variously reported to occur for some subjects under each of the different SO_2 exposure modes. The exposure of two subjects to several concentrations of SO_2 demonstrated clear dose-response relationships; that is, increases in SO_2 exposure levels resulted in increasingly larger SR_{aw} values with either oral or oronasal breathing for each subject. However, only one subject had a greater increase in SR_{aw} to oral inhalation of SO_2 at 0.5 and 1.0 ppm SO_2 than with oronasal exposure, whereas there were no differences seen between oral or oronasal exposures for the other subject even up to 2 ppm SO_2.

The results obtained by Kirkpatrick et al. (1982) using mouthpiece exposure to 0.5 ppm SO_2 are in agreement with those previously described by Sheppard et al. (1981b) and Linn et al. (1982a), in their pilot study, using the same exposure mode. When the results of these studies on asthmatic subjects are compared with results from studies of nonsensitive, healthy adults it appears that, with oral or oronasal breathing under moderate exercise conditions, asthmatic subjects are approximately an order of magnitude more sensitive to SO_2 exposure than nonsensitive, healthy adults. The results of the Kirkpatrick et al. (1982) and other studies of asthmatic subjects also demonstrate that specific exposure-effect relationships for SO_2-induced increases in bronchoconstriction are influenced by the level of exercise. For example, holding exercise levels constant at a moderately high level and using exposure to 0.5 ppm SO_2 via a face mask and/or mouthpiece, Kirkpatrick et al. (1982) demonstrated that the intensity of SO_2-induced bronchoconstriction effects in asthmatics varied as a function of mode of exposure in the following order: oral > oronasal > nasal. However, the observation by Kirkpatrick et al. (1982) of significant increases in SR_{aw} with facemask (oronasal) exposure to 0.5 ppm SO_2 differs from the results obtained by Linn et al., (1982a) in their study in which they exposed mild asthmatics to 0.5 ppm SO_2 during exercise ($V_e \cong 27$ ℓ/min) in an open chamber. This contrast in results is most likely due to the difference in exercise levels employed in the two studies, the exercise levels used in the Kirkpatrick et al. (1982) study resulting in ventilation rates (mean $V_e \cong 40$-44 ℓ/min) sufficiently high to ensure oronasal breathing with facemask exposure whereas the exercise levels in the Linn et al. (1982a) study were probably not sufficient to induce oronasal breathing during their open chamber exposure of exercising subjects. This suggests that significant increases in bronchoconstriction could be induced in asthmatic subjects with exposure to SO_2 levels below 1.0 ppm, if sufficiently high exercise levels were used to ensure a shift to oronasal breathing and, thereby, delivery of a greater proportion of inhaled SO_2 to tracheobronchial regions of the respiratory tract.

In an effort to assess this possibility, Linn et al. (1982b) effectively doubled (relative to their earlier study reported by Linn et al., 1982a) the SO_2 dose rate (concentration times ventilation) by exposing 23 young adult asthmatic subjects (21 to 27 years old) in an open chamber to 0.75 ppm SO_2 during moderately heavy exercise ($V_e \cong 40$ ℓ/min) for 10 min., once with unencumbered breathing and once under forced oral breathing conditions using noseclips and mouthpiece. Similar exposures to clean air alone, under identical temperature (23°C) and R.H. (90%) conditions, served as the control exposure condition. During clean-air exposures, SR_{aw} and symptoms increased significantly but with no meaningful differences seen between mouthpiece and unencumbered breathing. Exposures to 0.75 ppm SO_2 under these conditions produced significantly greater increases in SR_{aw} than clean-air exposures, regardless of the breathing mode (the SR_{aw} increases, however, being significantly greater with mouthpiece exposure than with unencumbered breathing). Symptom score changes and post-exposure forced expiratory function changes were qualitatively similar (i.e., little or no difference between unencumbered and mouthpiece breathing of clean air, increased symptom scores and a large decrement in expiratory function measures with unencumbered breathing of SO_2, and a large decrement in expiratory function measures with mouthpiece breathing of SO_2) to ΔSR_{aw} under SO_2 exposure conditions, but the excess responses seen with mouthpiece breathing did not attain statistical significance. These results of Linn et al. (1982b), like those of Kirkpatrick et al. (1982), demonstrated that mouthpiece breathing can compromise upper-respiratory defenses against SO_2 to the extent that respiratory function decrements are greater than or equal to those seen with oronasal breathing via chamber and facemask, respectively. In addition, the results of each study strongly reinforce each other and jointly demonstrate that SO_2-induced bronchoconstriction effects are possible at SO_2 levels below 1.0 ppm under exposure conditions which closely approximate the ambient situation during exercise.

The mechanisms by which bronchoconstriction is induced by SO_2 appear to include a neurally-mediated reflex, based on previous work by Nadel et al. (1965) and Sheppard et al. (1980). It has been hypothesized that release of chemical substances, eg. histamine, by degranulation of airway mast cells may also be indirectly involved in the mediation of the bronchoconstriction response. Sheppard et al. (1981a) evaluated this possibility by means of pharmacologic studies of the effects of disodium cromoglycate (cromolyn) on SO_2-induced bronchoconstriction. Disodium cromoglycate is known to inhibit the release of mediators from airway mast cells. In their study, Sheppard, Nadel and Boushey (1981a) evaluated SR_{aw} responses of six exercising asthmatics (who had marked bronchial hyperreactivity to inhaled histamine aerosol) to oral inhalation of 0.5 ppm (3 subjects) and 1.0 ppm (3 subjects) SO_2. Each subject was studied on three occasions, once breathing SO_2-free air, once breathing SO_2 with cromoglycate treatment, and a third test breathing SO_2 after a lactose placebo. Data were obtained before and after 10 minutes of exercise at a level inducing a minute ventilation of approximately 37-38 liters. In the cromolyn study, subjects inhaled 40 mg of cromolyn 20 minutes before the exercise and SO_2 exposure began. The effects of cromolyn alone were not determined. Statistical analyses were made by t-tests. Exercise alone did not increase SR_{aw}. However, SO_2 inhalation resulted in increases of SR_{aw} similar to the bronchoconstriction effects reported in the previous study by Sheppard et al (1980). Prior treatment with cromolyn significantly ($P < 0.025$) decreased SO_2-induced bronchoconstriction. This response was observed in all six subjects, although the dose utilized did not completely block bronchoconstriction in 2 of the 3 subjects breathing 1.0 ppm SO_2. No subjective symptomatic responses were reported. The results obtained support the view that SO_2 activates parasympathetic pathways indirectly by causing degranulation of mast cells and the consequent release of some chemical mediator such as histamine.

In another study, Koenig et al. (1982a) first exposed atopic adolescent subjects (having no clinical diagnosis of asthma) to either filtered air, 1 mg/m^3 NaCl droplet aerosol, 1 ppm SO_2, or 1 ppm SO_2 + NaCl aerosol for 30 minutes while at rest. No changes in pulmonary functions were observed as a consequence of exposures while at rest. (This is in contrast to observations made on extrinsic asthmatics exposed for 30 or 60 minutes while at rest in an earlier study by Koenig et al, 1980). Approximately 5-7 minutes later, the subjects in the present study walked on a treadmill at a level of exercise sufficient to increase their minute ventilation 5-6 times greater than their resting ventilation (absolute ventilatory volumes not reported). The subjects did not experience exercise-induced bronchospasm (EIB) following either of the sham exposures (i.e. air, NaCl droplet aerosol); however, in the presence of SO_2 (1 ppm) exercise-induced bronchospasm was observed in these atopic adolescents. The magnitude of the exercise-induced bronchospasm for SO_2 alone or SO_2 and NaCl droplet aerosol were the same. That is, oral inhalation of SO_2 or SO_2 + NaCl aerosol each produced essentially similar alterations in pulmonary functions: $FEV_{1.0}$ decreased by 24% ($P < 0.05$; paired t-Test); $V_{max\ 50\%}$ and $V_{max\ 75\%}$ were reduced by 29 and 34% respectively ($P < 0.05$; paired t-Test). While R_T (total airway resistance) increased significantly following the SO_2 + NaCl exposure, this measure of pulmonary function was not significantly altered following SO_2 exposure. No statistically significant pulmonary alterations were noted when these subjects exercised while breathing filtered air or NaCl aerosol alone. Koenig et al. (1982b) have also reported on the effects of SO_2 exposure of adolescents with no evidence of pulmonary disease. No significant changes in pulmonary functions were observed following exposure to SO_2 (1.0 ppm) or NaCl aerosol droplet alone while at rest. Only minor (but statistically significant) reductions in $FEV_{1.0}$, ($P < 0.025$; 3% decrease) were seen after exposure at rest to SO_2 (1.0 ppm) + NaCl aerosol droplet. Table 1 summarizes the data obtained by Koenig et al. (1981, 1982a, 1982b) on the three groups of adolescents they have studied. The data suggest that the degree of sensitivity to SO_2 depends on the relative magnitude of preexisting general hypersensitivity in the airways of human adolescents.

TABLE A-1. AVERAGE CHANGE (%) IN PULMONARY FUNCTION VALUES IN THREE GROUPS OF ADOLESCENT SUBJECTS AFTER EXPOSURE TO SO_2 (1 ppm) PLUS NaCl DROPLET AEROSOL (1 mg/m^3) DURING MODERATE EXERCISE (2-5 min. POST EXERCISE)

Pulmonary functional value	Extrinsic asthmatics	Atopics with EIB	Normals
R_T (3 Hz)	+67*	+41*	+3.0
$\dot{V}$max 50	-44*	-29*	-8.0*
$\dot{V}$max 75	-50*	-44*	-7.0
$FEV_{1.0}$	-23*	-18*	-6.0*
FRC	+7.0	+0.3	+10

* Statistically different from baseline.
Sources: Koenig, et al. (1981, 1982a, 1982b)

Previous work by Koenig et al. (1980, 1981) demonstrated that extrinsic adolescent asthmatics, unlike all normals, were sensitive to 1 ppm SO_2 in the presence of 1 mg/m^3 NaCl droplet aerosol under conditions of either rest or exercise via a mouthpiece. Although NaCl alone produced no such effects (decrease in $V_{max\ 50\%}$ + $V_{max\ 75\%}$), the lack of an "SO_2 alone" group made interpretation difficult.

The most recent study by Koenig et al. (1982a) described above on atopic non-asthmatic adolescents demonstrates that oral inhalation (mouthpiece) of either SO_2 or SO_2 + NaCl aerosol produced essentially similar alterations in pulmonary function parameters ($FEV_{1.0}$, $\dot{V}_{max}$ 50% and 75%, R_T) and affects both large and small airways. With these less sensitive adolescents, no changes in pulmonary functions were observed after exposure at rest, in contrast to observations (Koenig et al, 1980) made on extrinsic asthmatics at rest and exposed for 30-60 minutes to 1.0 ppm SO_2 plus 1mg/m^3 NaCl. The responses in atopic non-asthmatics were less in magnitude than those in a group of previously studied adolescent extrinsic asthmatics.

The studies by Koenig et al. (1980, 1981, 1982a,b) collectively demonstrate, in terms of pulmonary function parameters, that the sensitivity of adolescents to SO_2-induced bronchoconstriction decreases in the following order: extrinsic asthmatics > atopics > normals.

In summary, early studies indicated that some individuals in the presumed normal population were substantially more sensitive (i.e., hyperreacters) under controlled exposure conditions in regard to their responses (significant bronchospasm) to 1 ppm SO_2. These subjects were often noted to have histories of childhood asthma or of wheezing with viral upper respiratory infections. Despite these observations, systematic study of the effect of sulfur dioxide on clinically defined asthmatics was not initiated until 1980, when Sheppard et al. at the University of San Francisco and Koenig et al. at the University of Washington both reported that, in groups of asthmatic subjects, significant bronchoconstriction occurred on inhalation of 1.0 ppm sulfur dioxide. In some subjects the physiologic responses were accompanied by wheezing and shortness of breath. Subsequent studies by both of these research groups, using the same exposure mode (mouthpiece breathing), demonstrated that extrinsic asthmatics are more sensitive to 1 ppm SO_2, under exercising conditions ($V_e \sim 30$ ℓ/min) insufficient to induce bronchospasm. The Sheppard et al., (1980, 1981b) results and other studies (Kirkpatrick et al., 1982; Linn et al., 1982a, b) demonstrate that some asthmatic subjects are an order of magnitude more sensitive to SO_2 than nonsensitive, healthy adults. That is, whereas nonsensitive healthy adults display increased bronchoconstriction at 5 to 10 ppm while at rest and at levels possibly as low as 1 ppm with oral or oronasal breathing, clinically defined asthmatics appear to be more sensitive, as a group, down to 0.25 ppm SO_2 and the most sensitive (as individuals) down to

0.1 ppm under light to moderate exercise ($V_e \sim 30$ ℓ/min.) conditions. This potentiation of the effect of sulfur dioxide is attributed to both the greater dose (concentration times volume) and the rise in inspiratory flow that ultimately results in the penetration of more sulfur dioxide to the tracheobronchial region of the respiratory tract. Increased sensitivity to SO_2, less marked than in asthmatics, has also been observed among individuals without any signs of asthma (Koenig et al., 1982a; Stacy et al., 1981; Sheppard et al., 1980). These findings, that people with asthma and atopic disorders are more sensitive to sulfur dioxide and this sensitivity is further potentiated by mild exercise, are consistent with the theory that bronchial hyper-reactivity is associated with an increase in parasympathetically mediated reflex responses in the airways (Boushey et al. 1980).

In order to circumvent the criticism associated with direct extrapolation of the effects of SO_2 to the ambient situation in studies involving forced mouth (mouthpiece) breathing, additional experiments have been conducted by Nadel's group (Kirkpatrick et al., 1982) at the University of San Francisco and by Hackney's group (Linn et al., 1982a, 1982b) at the University of Southern California using different exposure modes. Both sets of studies demonstrate that mouthpiece breathing can compromise upper-respiratory defenses against SO_2 to the extent that respiratory decrements are greater than or equal to oronasal breathing via chamber and facemask, respectively. In addition, these results strongly reinforce each other with respect to the importance of exposure mode and exercise in inducing bronchoconstriction and jointly demonstrate that SO_2-induced bronchoconstriction effects and associated symptoms are possible under exposure conditions that closely approximate the ambient situation during exercise.

The health significance of the pulmonary function changes and symptomatic effects reported in the above studies is of importance in regard to decision-making related to the setting of standards for SO_2. Clear and indisputable resolution of what constitutes adverse health effects from among the effects demonstrated by these studies is probably not possible at this time. However, some important considerations can be stated which may assist in making reasonable and appropriate interpretations as to what the present results may imply regarding the potential or likely impact of SO_2 exposures on sensitive members of the general population under ambient conditions.

First, we should note that little controversy exists regarding the seriousness of full-fledged asthma attacks. That is, in the most extreme case, status asthmaticus, which occurs in as many as 10% of adults hospitalized for asthma (Senior and Lefrak, 1980), clearly represents a life-threatening medical condition. In less extreme cases of asthma attacks, also typified by airway constriction and various symptoms such as wheezing and dyspnea (but of lesser degree than is seen in cases of status asthmaticus) the day-to-day activities of affected individuals are often markedly disrupted or curtailed until medication is administered to relieve the symptoms and to ease their breathing.

In relation to the effects observed in the controlled human exposure studies discussed here, it should be emphasized that such studies are designed, in accord with currently accepted medical and research ethics, to avoid precipitating very serious asthmatic attacks or irreversible effects in exposed subjects. The question arises, then as to what the responses observed in the above studies may imply as far as being indications of potentially more serious effects among members of the general population exposed to SO_2 in ambient settings.

As stated in the Criteria Document, the temporary small changes in pulmonary function observed with SO_2 exposures of healthy ("normal") adults to ≤ 1.0 ppm SO_2 are of much less concern than the functional changes and symptoms observed in asthmatics in the present studies at SO_2 exposure levels below 1.0 ppm. Probably of most concern are the statistically significant increases in airway resistance and symptomatic effects (wheezing, dyspnea, etc.) observed: (1) with oral exposure to 0.5 ppm (1.3 mg/m^3) SO_2 during exercise (Sheppard et al. 1981b); (2) with oronasal exposure via facemask to the same SO_2 level during exercise (Kirkpatrick et al., 1982); or (3) with oronasal exposures to 0.75 ppm SO_2 during exercise in an open chamber most closely simulating likely ambient exposure conditions (Linn et al., 1982b). Such combined airway functional changes (bronchoconstriction) and symptomatic effects (wheezing, dyspnea, etc.) are likely to occur at 0.5-0.75 ppm SO_2 in the ambient air and are of concern in view of reports of indices of airway obstruction and presenting symptoms such as wheezing and dyspnea being among factors considered by physicians in determining the need for hospitalization of asthma patients following initial emergency room treatment (e.g., bronchodialator therapy) for asthmatic attacks.

The numbers of individuals in the general population potentially affected by SO_2 in terms of increased susceptibility to induction of airway constriction and for symptoms indicative of asthmatic attacks are difficult to estimate with precision, based on currently available data. However, individuals with non-asthmatic atopic disorders (e.g., hay fever, other allergies) make up 13.25% of the U.S. population in comparison to asthmatics (NIAID, 1979) that are estimated to comprise 4.5% (higher estimates have been made by Dodge and Burrows, 1980). Also, the undetected presence of asymptomatic atopic individuals in studies of presumed "normal" subjects may account for the recurrent finding of subjects "hyperreactive" to SO_2 who generally make up 10-20% of study groups evaluated in controlled human exposure studies of "normal" adults.

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GLOSSARY

Abiotic: Pertaining to the nonliving components of the environment, usually refers to a physical or chemical feature of the environment or ecosystem.

Absorption: Penetration of a substance into the bulk of a solid or liquid (cf. adsorption).

Accumulation mode: Particles formed principally by coagulation or growth through vapor condensation of short-lived particles in nuclei mode (see Aitken nuclei).

Acidic deposition: See Deposition.

Acidity: The quantity of hydrogen ions in solution; having a pH less than 7 (see pH).

Acute toxic effects: Effects of, relating to, or caused by a poison or toxin and having a sudden onset, sharp rise, and short course.

Adsorption: Solid, liquid, or gas molecules, atoms, or ions retained on the surface of a solid or liquid, as opposed to absorption, the penetration of a substance into the bulk of the solid or liquid.

Aerodynamic diameter: The diameter of a unit density sphere having the same settling speed (under gravity) as the particle in question of whatever shape and density.

Aerometry: Relating to measurement of the properties or contaminants of air.

Aerosol: A suspension of liquid or solid particles in a gas.

Aitken nuclei: Those particles and ions measured by means of an instrument in which water vapor is made to condense on particles by supersaturating the vapor; the term "condensation nuclei" is often used synonymously.

Atmospheric aerosols: A suspension in the atmosphere of microscopic particles of a liquid or a solid.

AISI light transmittance method: Technique for measuring ambient particulate matter by collecting the particles on a filter paper tape to determine the opacity of the stain expressed in terms of optical density or CoH units per 1000 feet of air sampled.

AISI tape sampler: See AISI light transmittance method.

Alkalinity: The quantity of hydroxide ions in solution; having a pH greater than 7 (see pH).

Anion: A negatively charged ion.

Anthropogenic emissions: Emissions resulting from the impact of human activities on the natural world.

Artifact: 1. A structure in a fixed cell or tissue formed by manipulation or by the reagent. 2. An erroneous estimate of the atmospheric concentration of a gaseous or particulate species due to chemical or physical modification during sampling, storage, or analysis. 3. A structure or substance not normally present, but produced by some external agency or action.

Atmospheric aerosols: A suspension in the atmosphere of microscopic particles of a liquid or a solid.

Atmospheric turbidity: Any condition of the atmosphere that reduces its transparency to radiation, especially to visible radiation. Cloudy or hazy appearance in an atmosphere caused by a suspension of colloidal liquid droplets or fine solids.

Benthic macroscopic plants: Flora and fauna large enough to be observed by the naked eye occurring on the bottom underlying a body of water.

Beta attenuation analysis: A method of estimating mass concentrations of particles by using the differential attenuation of electrons.

Biogenic: Produced by actions of living organisms.

Bioindicator: Any species of plant or animal that is particularly sensitive to a specific pollutant.

Biomass: The total amount of living organic matter in a given ecosystem, usually expressed as dry weight per unit area.

Biosphere: The portion of the earth in which living systems are encountered, including the lower part of the atmosphere, the hydrosphere, and the lithosphere to a depth of about 2 kilometers.

Biota: Pertaining to the living systems of the environment, animals, plants, and microorganisms.

Blue sky scattering: See Rayleigh scattering.

British Smokeshade (BS) sampler: Device used to measure the reflectance of particles collected on a filter and to predict mass concentrations.

Bronchoconstriction: Constriction relative to or associated with the bronchi or their ramifications in the lungs.

Bronchospasm: Temporary narrowing of the bronchi due to violent, involuntary contraction of the smooth muscle of the bronchi.

Carbachol ($C6H_{15}\ ClN_2O_2$): The choline ester, carbamycholine chloride, used principally as a miotic (pupil constrictor) in the local treatment of glaucoma and as a bronchoconstrictor.

Carcinogenesis: The production of cancer.

Cardiorespiratory effects: Influence of a substance on the functioning of the heart and lungs.

Cascade impactors: A device for sampling an aerosol that consists of sets of jets of progressively smaller size and collection plates designed so that each plate collects particles of one size range.

Catchment basin: The geological structure of a lake or stream.

Cation: A positively charged ion.

Chemoreceptor: Any sensory organ that responds to chemical stimuli.

Chemiluminescence: Emission of light as a result of a chemical reaction without an apparent change in temperature. Used in determining concentration of some pollutant gases.

Chlorosis: A disease condition of green plants seen as yellowing of green parts of the plant.

Chronic toxic effects: Characterized by a slow progressive course of toxicity of indefinite duration.

Ciliary beat frequency: Rate of pulsation of the minute vibratile, hairlike processes attached to the cells lining some airways.

Cloud: A free aerodisperse system of any type having a definite form and without regard to particle size.

Coarse particles: Airborne particles larger than 2 to 3 micrometers (μm) in diameter.

Coefficient of haze (CoH): Measurement of the optical density of a sample of suspended particulates collected by the AISI light transmittance methods.

Cohort: A group of individuals or vital statistics about them having a statistical factor in common in a demographic study (as year of birth).

CoH: See Coefficient of haze.

Colorimetry method: Chemical analysis in which the amount of a chemical substance present is found by measuring the light absorption due to its intrinsic color or the color of another substance into which it can be completely converted. Used in determining presence of atmospheric SO_2.

Condensation nuclei: See Aitken nuclei.

Condensed organic vapors: See Polycyclic organic matter.

Coulometry: A chemical technique for measuring average current strength.

Critical damage point: The point at which the service life or utility of the material ends or is severely impaired.

Crop monoculture: The agricultural practice of growing a single crop species. Opposite of a natural ecosystem in which a wide variety of flora and fauna interact.

Cultivar: A cultivated variety or species of crop plants. Abbreviated cv. Also known as cultigen.

Cyclone samplers: A centrifugal device for separating particles from an aerosol.

Deposition:

Acidic--Removal of acidic pollutants from the atmosphere by dry and wet deposition.

Dry--Removal of pollutants from the atmosphere through interactions with various surfaces of plants, land, and water.

Respiratory tract--Removal of inhaled particles by the respiratory tract which depends on breathing patterns, airway geometry, and the physical and chemical properties of the inhaled particles.

Wet--Removal of pollutants from the atmosphere by precipitation.

Dicarboxylic acids: Compounds with two carboxyl groups.

Dichotomous sampler: A device used to collect separately fine and coarse particles from an aerosol.

Dust: Dispersion aerosols with solid particles formed by comminution or disintegration, without regard to particle size.

Ecosystem: A functional unit of the environment that includes all organisms and physical features within a given area. Derived from ecological system.

Aquatic--An ecosystem functioning in a marine environment.

Terrestrial--An ecosystem functioning on the land surface of the earth.

Edaphic factors: Factor of or relating to the soil.

Electrical Aerosol Analyzer (EAA): A device for measuring the size distribution of particles of 0.01 to about 1.0 μm diameter. The particles pick up electric charges according to their size and are then analyzed by electrostatic precipitation and an electrometer.

Electroencephalogram alpha-rhythms: Alpha waves graphically depicted on an electroencephalogram.

Epithelium: A primary animal tissue, distinguished by closely packed cells with little intercellular substance; covers free surfaces and lines body cavities and ducts, such as in the respiratory tract.

Expiratory flowrate: See Pulmonary measurements.

Fine particles: Airborne particles smaller than 2 to 3 micrometers in diameter.

Flame photometric detection: A process by which a spray of metallic salts in solution is vaporized in a very hot flame and subjected to quantitative analysis by measuring the intensities of the spectrum lengths of the metals present.

Fluorescence analysis: A method of chemical analysis in which a sample, exposed to radiation of one wavelength, absorbs this radiation and reemits radiation of the same or longer wavelength in about 10^{-9} second. The intensity of reemitted radiation is almost directly proportional to the concentration of the fluorescing material. Also known as fluorometry.

Fogs: Suspension of liquid droplets formed by condensation of vapor or atomization; the concentration of particles is sufficiently high to obscure visibility.

Foliar uptake: Uptake through the leaves of plants.

Fugitive emissions: Air pollutants arising from human activities, such as roadway and industrial dust, that do not emanate from a particular point, such as an exhaust pipe or stack, and are not readily amenable to control.

Fumes: Condensation aerosols containing liquid or solid particles formed by condensation of vapors produced by chemical action of gases or sublimation.

FVC: The volume of air that can be forcibly expelled from the lungs after the deepest inspiration.

Glycoprotein synthesis: The creation of a class of conjugated proteins containing both carbohydrate and protein units.

Gravimetric mass method: Measurement technique in which the amount of the constituents is determined by weighing.

Gravimetry: Measurement of a weight or density.

Haze: An aerosol that impedes vision and may consist of a combination of water droplets, pollutants, and dust.

Hematology: The science of the blood; its nature, functions, and diseases.

High volume (hi-vol) sampler: A high flow-rate device used to collect particles from the atmosphere.

Hilar lymph nodes: Nodes located in that part of a gland or of certain organs, especially the lung, where the blood vessels, nerves, or ducts leave and enter.

Hydrogen peroxide method: A titrimetric method for providing aerometric SO_2 estimates.

Hydroxyl radical: Chemical prefix indicative of the [OH] group.

Hygroscopic growth: Growth induced by moisture.

IFR (Instrument Flight Rules) instrumentation: System put into effect by Federal Aviation Administration which restricts flight in controlled air spaces when visibility falls below 4.8 kilometers, causing the grounding of most small aircraft.

Integrating nephelometer: See Nephelometry.

Intratracheal instillation: Process of placing material within or through the trachea.

Ion exchange chromatography: A chromatographic procedure in which the stationary phase consists of ion-exchange resins which may be acidic or basic.

Irritant potency: The relative strength of an agent that produces irritation.

Isopleth: 1. A line of equal or constant value of a given quantity with respect to either space or time. Also known as an isogram; 2. A line drawn through points on a graph at which a given quantity has the same numerical value as a function of the two coordinate variables.

Koschmieder relationship: The inverse proportionality between visual range and total extinction.

LC_{50}: Concentration of a substance lethal to 50 percent of tested species.

Leach: 1. The dissolving, by a liquid solvent, of soluble material from its mixture with an insoluble solid; 2. The separation or dissolving out of soluble constituents from a rock or ore body by percolation of water; 3. Dissolving soluble minerals or metals out of the ore, as by the use of percolative solutions, such as cyamide or chlorine solutions, acids, or water. Also known as lixiviation.

Linear model: A model where all the interrelationships among the quantities involved are expressed by linear equations which may be algebraic, differential, or integral.

μm: Micrometer.

Mechanical clearance: See Mucociliary action.

MEFR: *See* Pulmonary measurement.

Megalopolis: 1. A very large city; 2. A thickly populated region centering on a metropolis.

Methylthymol blue method: Technique for measuring suspended sulfates and sulfuric acid involving a collection of samples on sulfate-free glass fiber filters by high-volume particulate matter samplers.

Middle turbinate region: Area encompassed by the *concha nasalis media ossea*.

Minute volume (Ve): *See* Pulmonary measurements.

Mist: Suspension of liquid droplets formed by condensation of vapor or atomization; the droplet diameters exceed 10 μm and in general the concentration of particles is not high enough to obscure visibility.

MMFR: *See* Pulmonary measurements.

Morbidity: 1. The quantity or state of being diseased; 2. The ratio of the number of sick individuals to the total population of a community.

Morphology: Structure and form of an organism at any stage of its life history.

Mortality rate: For a given period of time, the ratio of the number of deaths occurring per 1000 population. Also known as death rate.

Mucociliary action: Ciliary action of the mucous membranes lining the airway that aids in cleansing and removing irritants and aids in moving particles to the pharyngeal regions.

Mutagenesis: An abrupt change in the genotype of an organism, not resulting from recombinations; genetic material may undergo qualitative or quantitative alteration, or rearrangement.

Nasopharyngeal absorption: The taking up of fluids, gases, or particles by and within the nasopharynx.

Necrotic lesions: A cell or group of cells undergoing necrosis (i.e., dying as a result of injury, disease, or other pathologic state).

Nephelometry: 1. The study of aerosols using the techniques of light scattering. 2. Measurement of light scattering coefficient by certain optical instruments.

Oncogenesis: Process of tumor formation.

Optical density: The degree of opacity of a translucent medium expressed by log I_o/I, where I_o is the intensity of the incident ray, and I is the intensity of the transmitted ray, abbreviated OD.

Optical particle morphology method: Techniques for identifying the character and sources of collected particles.

Oronasal breathing: Breathing through the nose and mouth.

Osmoregulation: A physiological regulatory mechanism for the maintenance of an optimal and constant level of osmotic activity of the fluid in and around the cells.

Oxidation (various types): A chemical reaction in which a compound or radical loses electrons 1/m that is, in which the positive valence is increased.

Pararosaniline method: Manual method for determining the concentration of atmospheric SO_2.

Particle: Any object, solid or liquid, having definite physical boundaries in all directions; in air pollution, practical interest concentrates on particles less than 1 mm in diameter.

Particulate matter (PM): Matter in the form of small airborne liquid or solid particles.

Pathogen: A disease-producing agent; usually refers to living organisms.

Personnel dosimeter sampling: Determination of the degree of exposure on individuals, using survey meters, and determination of the dose received by means of dosimeters.

pH: A measure of the effective acidity or alkalinity of a solution. It is expressed as the negative logarithm of the hydrogen-ion concentration. Pure water has a hydrogen ion concentration equal to 10^{-7} moles per liter at standard conditions (25°C). The negative logarithm of this quantity is 7. Thus, pure water has a pH value of 7 (neutral). The pH scale is usually considered as extending for 0 to 14. A pH less than 7 denotes acidity; more than 7, alkalinity.

Phagocytosis: A mechanism by which macrophages engulf and carry away particles.

Pharyngeal regions: The chamber at the oral end of the vertebrate alimentary canal, leading to the esophagus.

Photochemistry: The study of the effects of light on chemical reactions.

Physical damage functions: The mathematical expression linking exposure to damage, expressed in terms appropriate to the interaction of the pollutant and material.

Planetary boundary layer: First layer of the atmosphere extending hundreds of meters from the earth's surface to the geostrophic wind level, including, therefore, the surface boundary layer and the Ekman layer; above this layer lies the free atmosphere.

Polycyclic organic matter: Compounds including both polycyclic aromatic hydrocarbons (homocyclics) and heterocyclic analogs, having low vapor pressure and usually condensed on the surface of fine particles in the atmosphere. Abbreviated POM.

Potentiation: The combined action of two drugs, greater than the sum of the effects of each used alone.

Primary particles (or primary aerosols): Dispersion aerosols formed from particles that are emitted directly into the air that do not change form in the atmosphere.

Pulmonary Measurements: Measurements of the volume of air moved during a normal or forced inspiration or expiration, which is a reflection of pulmonary compliance. Atmospheric pollutants can seriously impair the volumes of air/gas exchanged during the ventilatory function. Specific lung volume measurements include:

Tidal volume (TV)--The volume of air moved during normal inspiration.

Functional residual capacity (FRC)--The amount of air left in the lung at the end of a normal expiration.

Expiratory reserve volume (ERV)--Air removed from the lung by forced expiration.

Residual volume (RV)--Air that cannot be expelled from the lung.

Vital capacity--The sum of ERV, TV, and inspirational reserve volume (IRV).

Rales: An abnormal sound accompanying the normal sounds of respiration within the air passages and heard on auscultation of the chest.

Rayleigh scattering: Scattering of electromagnetic radiation by bodies much smaller than the wavelength of the radiation. For visible wavelengths, the molecules constituting the atmosphere cause Rayleigh scattering.

Refractive index method: The ratio of the phase velocity of light in a vacuum to that in a specified medium. Also known as refractive index; refracture index.

RH (Relative Humidity): The dimensionless ratio of the actual vapor pressure of water in the air to the saturation vapor pressure.

Secondary particles (or secondary aerosols): Dispersion aerosols that form in the atmosphere as a result of chemical reactions, often involving gases.

Smaze: A combination of "smoke" and "haze".

Smog: A combination of "smoke" and "fog". Originally, this term referred to episodes in Great Britain that were attributed to coal burning during persistent foggy conditions. In the United States, "smog" has become associated with urban aerosol formation during periods of high oxidant concentrations.

Smoke: Dispersion aerosol containing both liquid and solid particles formed by condensation from supersaturated vapors.

Spectrometry, second-derivative: A technique for measuring ambient SO_2.

Spirometry: The measurement, by a form of gas meter (spirometer), of volumes of air that can be moved in and out of the lungs.

Stomata: Plural of stoma. Any minute pore, orifice, or opening on a free surface; specifically, one of the openings between epithelial cells of a lymph space.

Sulfate: 1. A compound containing the [SO_4^{2-}] group, as in sodium sulfate (Na_2SO_4); 2. A salt of sulfuric acid.

Sulfation methods: Tests used to estimate ambient SO_2 concentrations over extended time periods.

Sulfur dioxide (SO_2): A toxic, irritating, colorless gas; soluble in water, alcohol, and ether; boils at -10°C; used as a chemical intermediate in paper pulping, a solvent, a disinfectant, and a preservative; emitted by the combustion of sulfur-bearing fuels. Also known as sulfurous acid anhydride.

Sulfur oxides: Oxides of sulfur, such as sulfur dioxide (SO_2) and sulfur trioxide (SO_3).

Synergism: The joint action of agents so that their combined effect is greater than the algebraic sum of their individual effects.

Systemic: Pertaining to or affecting the body as a whole.

Telephotometry: Measurement of the apparent brightness of distant objects.

Thoracic: Of or pertaining to the chest.

Thorax: The chest.

Tidal volume (TV): See Pulmonary measurements.

Tracheobronchial region: The area encompassed by the trachea and bronchi.

Transmissometry: The technique of determining the extinction characteristics of a medium by measuring the transmission of a light beam of known initial intensity directed through that medium.

Troposphere: Free tropospheric exchange: The portion of the atmosphere from the earth's surface to the tropopause; that is, the lowest 10 to 20 kilometers of the atmosphere.

Tumorigenesis: Formation of tumors.

Turbidimetry: A scattered-light procedure for the determination of the weight concentration of particles in cloudy, dull, or muddy solutions; uses a device that measures the loss in intensity of a light beam as it passes through the solution.

Visual range: The maximum distance at which a large black object can be seen against the horizon sky in daytime.

West-Gaeke method: See Pararosaniline method.

X-ray fluorescence: Emission by a substance of its characteristic X-ray line spectrum upon exposure to X-rays. Also known as X-ray emission.